Journal
OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

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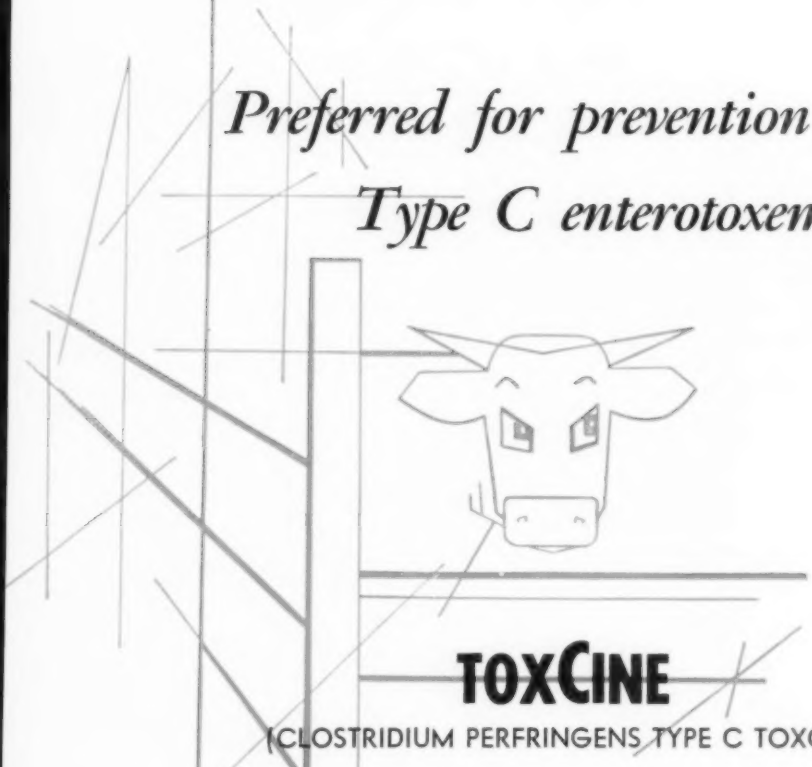

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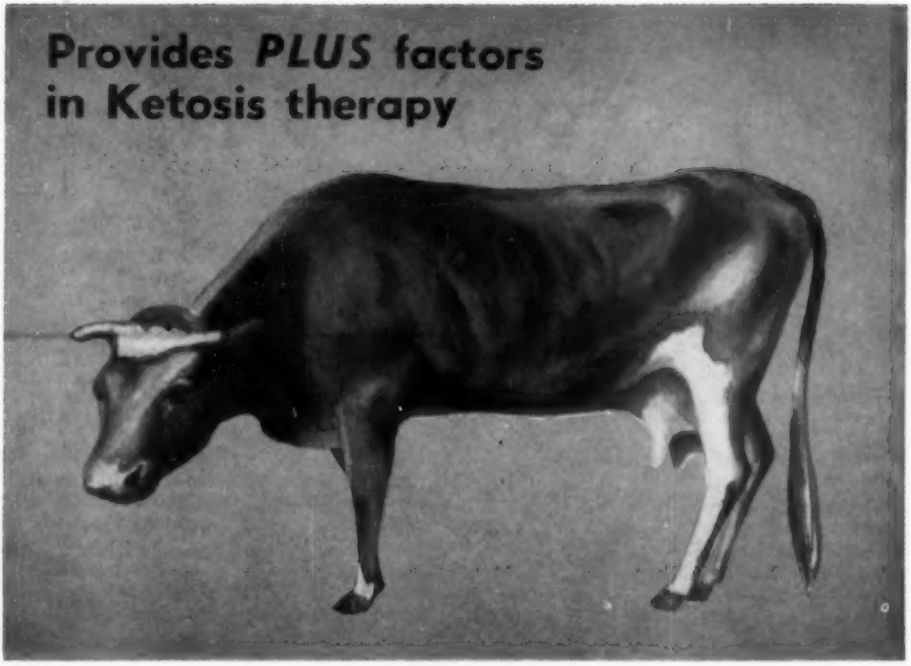

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Journal of the American Veterinary Medical Association

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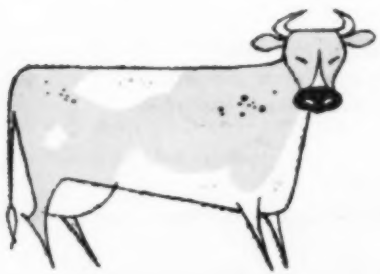

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Announcement

Revision of the premium for

PROFESSIONAL LIABILITY INSURANCE

for AVMA Members

After reviewing the experience of the last few years the National Bureau of Casualty Underwriters (the official rate-making organization) obtained the approval of most State Insurance Departments to the following annual premium charges for Veterinarians' Liability Insurance.

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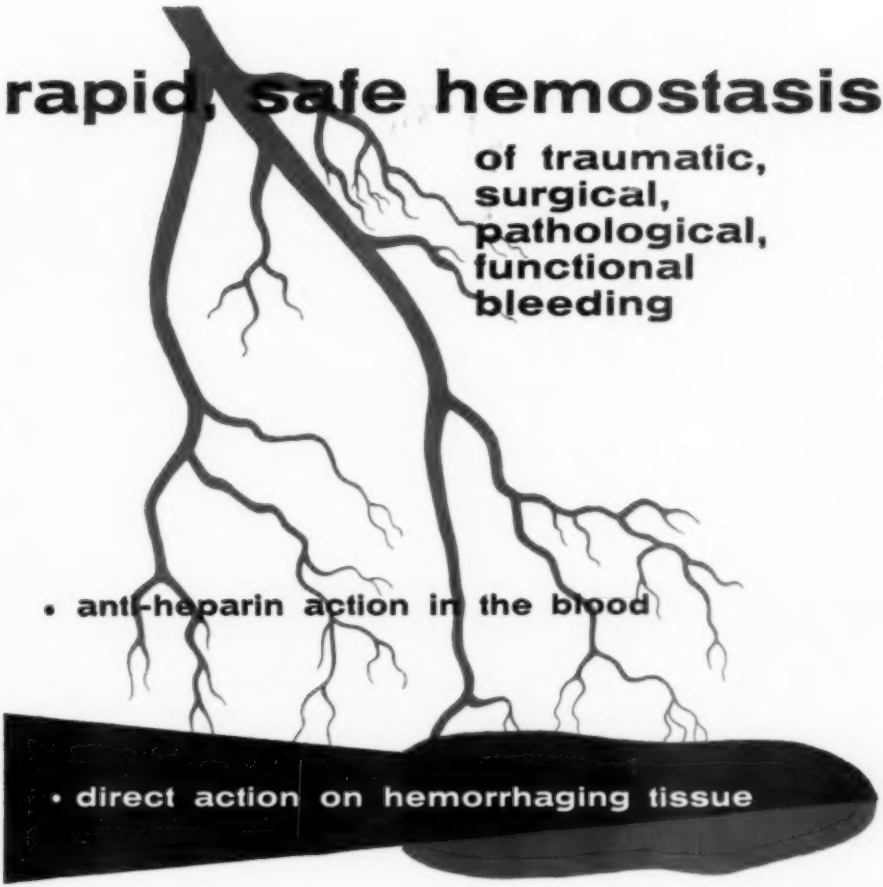

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AVMA ☆ Report

AVMA Adopts Health and Accident Insurance Plan

The House of Representatives, in session during the San Antonio AVMA Annual meeting, approved the report of the AVMA Special Committee on Insurance, which contained recommendations of immediate and vital interest to AVMA members.

This action by the House establishes an insurance program for AVMA members that has a number of unique features.

a) The insurance is on a **true group** basis, with a much higher percentage of the premiums to be returned in benefits and cash dividends than is possible with individual policies or the "franchise" type plans now in force for nearly all other professional association plans.

b) This program will be administered by an AVMA Group Insurance Trust, and not by the insurance company. The trustees will consist of six (6) AVMA officers (Executive Board chairman, president, president-elect, immediate past-president, executive secretary, and treasurer), and three members elected by the AVMA Executive Board.

c) It is the first professional association group plan to provide long-term catastrophe income insurance for members disabled by accidents or sickness, plus catastrophe hospital and surgical insurance for members and their dependents.

d) It is the first professional association group to include true group life insurance in a comprehensive program of personal protection with accident and sickness insurance.

e) It is the first time that one of the ten largest mutual life insurance companies has underwritten a comprehensive group program for any professional association.

The first basic coverage in the AVMA insurance plan is **disability income insurance** for members disabled by accidents or sickness.

This program is specifically designed to meet the hazards of prolonged injury or illness. Monthly income benefits are payable up to **five years** for such disabilities caused by sickness and up to **life time** for those caused by accident. Two types of plans will be offered:

Plan I provides benefits beginning on the first day for accidents and the eighth day for sickness or the first day of hospitalization, whichever occurs first.

Plan II provides benefits beginning on the 31st day for both accidents and sickness with reduced premiums.

The second basic coverage, combined with the monthly income insurance, is a plan of true **group life insurance.**

The amount of insurance coverage varies with age, being highest in the earlier years when the need for life insurance is greatest, and reducing each year to age 65.

The premium rate per $1,000 of life insurance is extremely low. At age 25 it amounts to only $3.33 per year; at age 35 it is only $5.00 per year; at age 45 it is only $10.00 per year; at age 55, $20.00 per year, and at age 65 only $44.00 per year.

The third basic feature is accident insurance; a principal sum bene-

(Continued on adv. p. 26)

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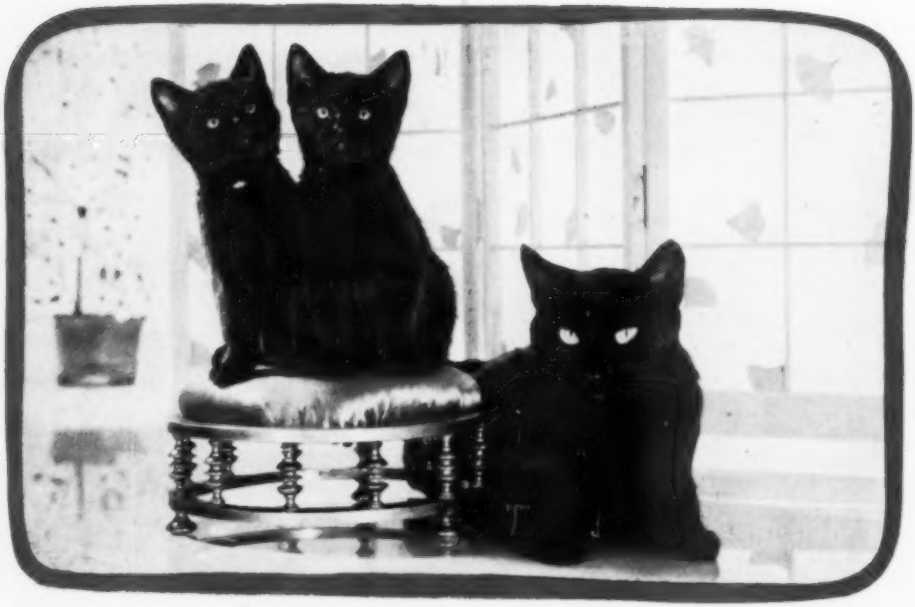

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- ☐ folder N-4 syringe cases
- ☐ folder N-5 sign letters
- ☐ folder N-6 firing irons
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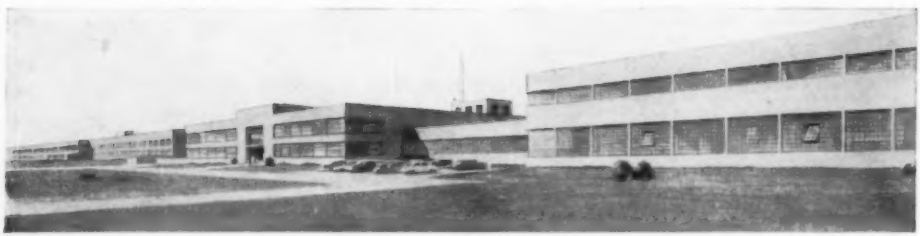

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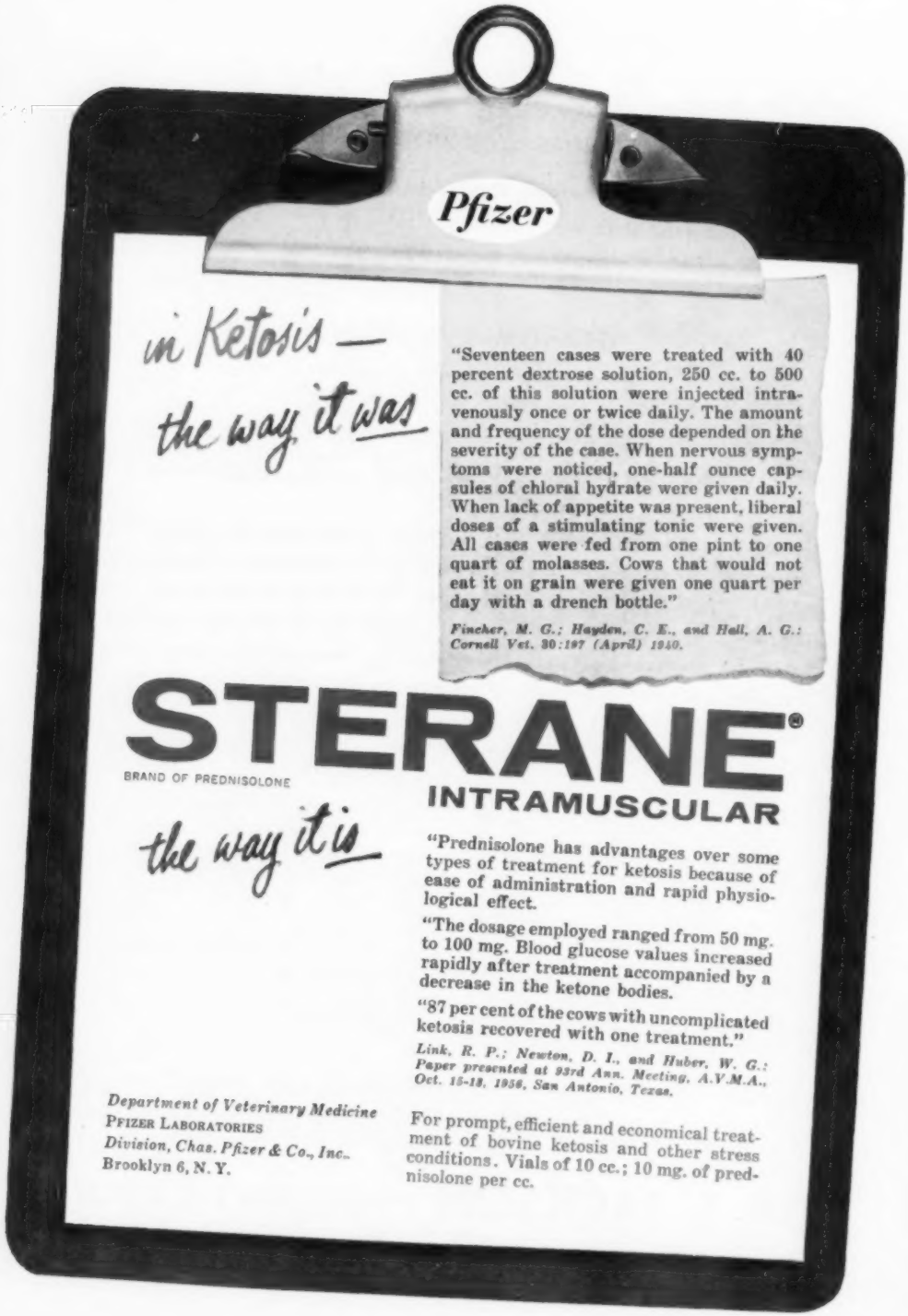
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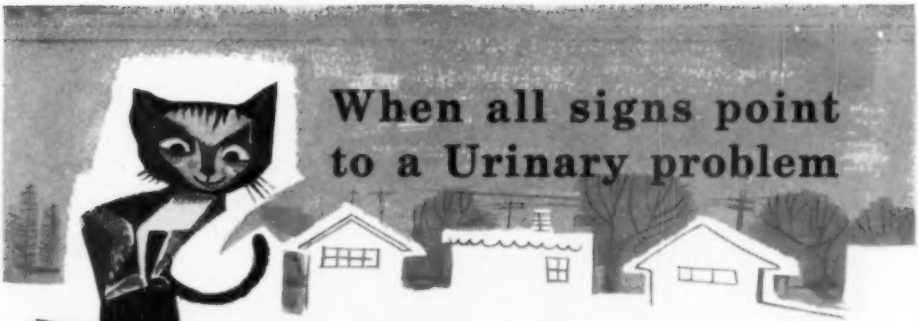

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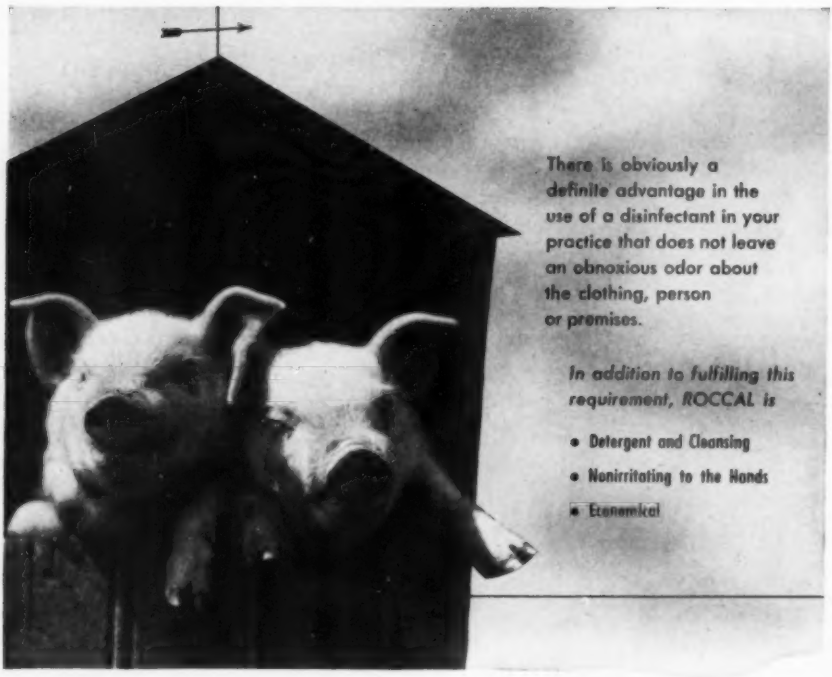
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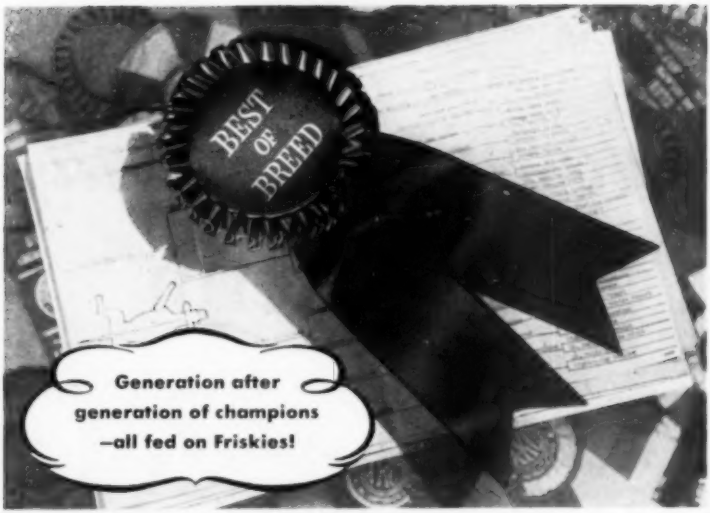

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Further Evaluation of the Treatment of Disc Protrusion Paraplegia in the Dog

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Auburn, Alabama

IT HAS BEEN established that a protrusion, herniation, or rupture of an intervertebral disc in the dog can cause one or more of the following symptom complexes: pain, paraplegia (spastic, flaccid), paresis, and acute progressive paralysis. The first three conditions tend to cause a focal myelitis or a focal pachymeningitis, or both, from pressure of the disc on the cord. The latter condition is a diffuse, progressive, hemorrhagic inflammation of the cord and terminates in respiratory paralysis and death.

The outcome of the painful, paretic, and paraplegic conditions without radical treatment depends largely on the location and extent of the lesion in the spinal cord, as well as the animal's ability to compensate neurologically for the focal presure on the nerve elements. Since some animals recover spontaneously, it is difficult to evaluate therapy. However, the condition frequently recurs and each recurrence produces more marked and permanent changes which require more radical therapeutic efforts.

Previous work by surgeons, both in human and veterinary medicine, has demonstrated the value of surgical intervention as a treatment for disc protrusions. This paper will present data obtained from long-term observations on disc disorders treated by conservative and surgical methods.

DIAGNOSIS

Diagnosis[1,2] of disc protrusion paraplegia can usually be made easily if seen in the susceptible breed and usual age range. The physical signs include a characteristic, superficial pain area on the dorsum of the back. Spinograms may be needed for a differential diagnosis from other possible spinal conditions. A narrow disc space, with or without showing calcium, is significant if it is in the area of the superficial pain. A calcified protrusion or intervertebral foramen in or adjacent to a pain area is also significant. If no lesions are seen or if the above are not correlated with a superficial pain area, then contrast myelography should be performed, but only if surgery is anticipated.[2] If conservative treatment is to be employed, contrast mediums should not be used because of their slight irritating effect on the meninges.[2]

Data as to the occurrence, breed and age incidence, symptomatology, and diagnosis of intervertebral disc protrusions in the dog have already been presented.[1-7] However, information on methods of treatment will be repeated briefly, especially that on newer drugs or modifications in techniques.

CONSERVATIVE TREATMENT

Generally, conservative treatment may be employed in the following cases: (1) an animal with pain but no marked paresis or paraplegia; (2) one which does not have loss of support, has normal reflexes, and which defecates and micturates normally (however, such cases should be watched closely for regression); (3) one which has diffuse multiple lesions compressing the cord; and (4) one with acute disc protrusion with progressive ascending paralysis.

Conservative treatment largely involves symptomatic care and good nursing. Rest and maintenance of urine and fecal elimination are imperative. Hot water baths, with passive exercise or massage adminis-

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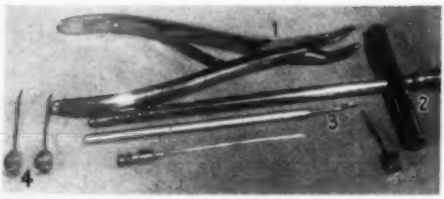

Fig. 1—Instruments used in disc surgery: (1) dean mastoid rongeur, (2) michele trephine, 7/32 inch, (3) claw tartar scraper (ground to approximately 1/16 in. width), and (4) 14- to 16-gauge intravenous needles.

tered twice daily for ten to 15 minutes each, are helpful in relieving muscle spasms, in preventing muscle atrophy and the formation of decubital ulcers, and in stimulating general recuperation. Diathermy has been used and ultrasonic applications[8] are being evaluated. Good nutrition with parenteral fluid administration, if needed, is indicated.

Various nerve and metabolic stimulants, such as B complex vitamins, physostigmine, calcium, thyroid, and parathyroid, may be helpful. Sedatives and antispasmodic agents, such as barbiturates and mephenesin, have been used. Anodyne and anti-inflammatory drugs, such as salicylates, cinchophens, butazolidin, and adrenocortical steroids, have received attention.[6,7,9,10]

The anti-inflammatory drugs and anodynes are therapeutically sound only when combined with enforced rest. If these drugs are given without such confinement, the dog may reinjure itself more severely because of a sense of well-being created by the drug. The metabolic stimulants are worthwhile after the acute condition has subsided, although most animals apparently respond without much medication as long as proper nursing care and restricted exercise are continued.

Cystitis, one of the most frequent sequelae in any paraplegia, results from the inability to urinate properly and may develop into a severe hemorrhagic form. The parenteral use of broad-spectrum antibiotics, together with instillation of antibiotics and topical coagulants (if hemorrhagic) into the bladder, are indicated.

SURGICAL TREATMENT

Surgery should be employed as soon as possible after the injury—within three to five days if paralysis and allied neuropathies are profound. The pressure in these cases will cause an irreversible damage unless relieved soon. In general, surgery may be considered in the following instances: (1) when recurrences are progressively worse; (2) when there is profound paralysis, with loss of urinary and defecatory functions; and (3) when there is no response to conservative treatment.[10]

The surgical correction of disc protrusion involves two recommended procedures —disc fenestration or curettage, and hemilaminectomy. Both procedures should be performed on the pathological disc for proper and complete correction.

Since these procedures have been previously described in detail,[4,10,11,12] the author's recommended procedure will be briefly outlined.

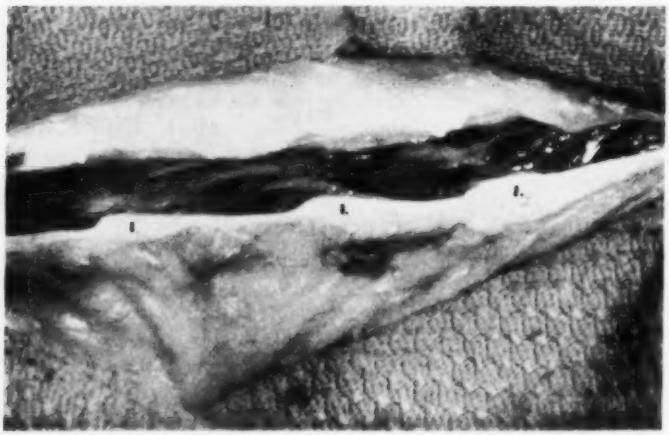

Fig. 2—After the skin is incised, the dorsal lumbar fascia is incised just lateral to the spinous processes; (1) spines.

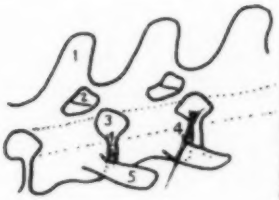

Fig. 3—The musculature is bluntly dissected from the vertebrae; (1) spinous process, (2) articular process, (3) intervertebral foramen, (4) dorsal spinal nerve, and (5) lateral process.

1) An incision is made on the dorsal midline, over or just lateral to the spinous processes (fig. 1, 2).

2) After incising the dorsal lumbar fascia, the musculature is removed by blunt dissection from the lateral surface of the spines and bodies of the vertebrae down to the lateral processes (fig. 3).

3) The tendon attachments are carefully removed from the articular and accessory processes.

4) The dorsal lumbar nerves and vessels are identified as they emerge from the intervertebral foramen and are avoided.

5) Curettage.—The intervertebral disc is located just anterior to the junction of the lateral process to the vertebral body (fig. 4) and is curetted with a 14- or 16-gauge, long-beveled intravenous needle or a hook-type dental tartar scraper which has been ground to a narrower width. The instrument is directed through the disc toward

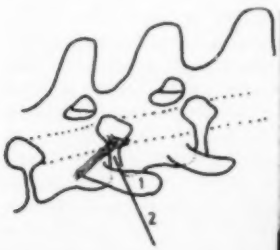

Fig. 4—A small-gauge needle is used to locate the disc just anterior to the junction of the lateral process to the body of the vertebra; (1) lateral process, and (2) needle in intervertebral disc.

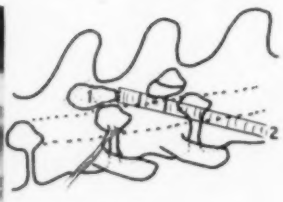

Fig. 5—Trephination of the lateral lamina after the articular process has been clipped off smooth with the rest of the vertebra; (1) articular process, and (2) michele trephine.

the opposite side and as much of the disc as possible is removed. Adjacent discs may be curetted through the same incision for prophylactic measures, especially if surgery is at the thoracolumbar junction. Properly curetted discs will give no future trouble.

6) Hemilaminectomy.—Hemilaminectomy consists of removing a portion of the lateral bony covering of the spinal canal in order to fully remove the disc from the top side and to allow room for expansion of the cord. It may be done by chiseling or by nipping off bone with a rongeur forceps, although a 5/16-inch laminectomy trephine is the preferred instrument. The articular process is removed and a trephine opening is carefully made at this point (fig. 5, 6). This opening can be made larger with mastoid rongeur forceps (fig. 7). The necrotic disc mass is removed with small mosquito forceps and curettage is completed by use of the needle or dental scraper (fig. 8).

Fig. 6—During trephination, the area is frequently checked for depth and looseness.

7) A solution containing a topical coagulant and an antibiotic is applied to the area. A section of absorbable gelatin sponge (gelfoam)* is placed over the bony defect (fig. 9). The dorsal lumbar fascia is securely sutured and a routine skin closure is made.

In performing the above surgery, hemostasis is imperative. If "oozing" hemorrhage from the muscle or bone occurs, packing and patience is required. Occasionally, topical and parenteral coagulants are helpful. In the terminal thoracic region, the disc lies about ⅛ to ¼ inch anterior to the junction of the rib to the body of the vertebra. Here, the curet must be directed laterally and not ventrally since the aorta and vena cava lie ventral to the vertebral bodies.

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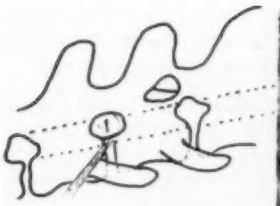

Fig. 7—The trephine opening (1) which has exposed the dura mater of the spinal cord.

An outline for the surgical correction of cervical disc protrusions follows.

1) Make an incision on the ventral midline of the animal's neck.

2) Carefully dissect through the musculature, then retract the trachea, esophagus, large vessels, and nerves laterally (fig. 10).

3) Dissect the musculature from the affected disc and adjacent prominences on the ventral midline of the vertebral column.

4) Curet the disc as far upward (dorsally) as possible from the ventral approach (hemilaminectomy in this area is difficult) using the needle or tartar scraper (fig. 11). The proper curettage distance can be ascertained by introducing a small-gauge injection needle with a dulled point into the disc and directing it dorsally until a flinch or twitching sensation is obtained. This sensation is due to the pressure of the needle on the dura mater.

5) The musculature should be loosely sutured; close the subcutaneous fascia and skin routinely.

The chief difficulty in cervical disc surgery is the location of the offending disc since the landmarks are obscure, especially with the animal in the dorsal recumbent position. A "spot" radiograph during surgery is helpful.

The surgical aftercare is of the utmost importance. All the nursing procedures discussed in the section on conservative treatment are indicated. Generally, there is considerable postsurgical pain, which

Fig. 8—The curetted disc (1) seen through the hemilaminectomy opening.

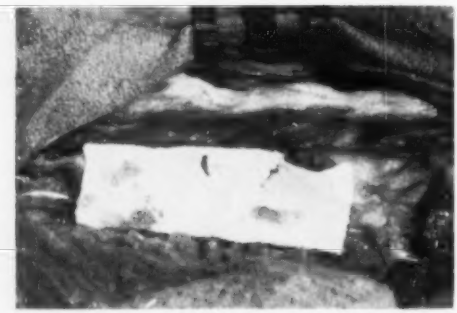

Fig. 9—Absorbable gelatin sponge material is placed over the bony defect.

Fig. 10—Ventrolateral view. In cervical disc protrusions, the incision is made on the ventral midline. The trachea (1), esophagus, and large vessels and nerves are retracted to one side.

makes the animal reluctant to move. This pain is a good "governor" of activity and should not be relieved unless it is excessive. Urine and fecal elimination should be maintained faithfully and broad-spectrum antibiotics administered for three to four days postsurgically to curb cystitis. If hemostasis was not complete during surgery, serum may accumulate and should be aspirated aseptically. A compression bandage is helpful in preventing further fluid accumulation.

Hydrotherapy, with hot water, and supervised exercises are initiated two to three days after surgery. A plastic or collodion dressing applied to the incision will seal it from the water. Initially, the dog is placed on its feet and is encouraged to stand. Gentle pressure on the flank areas may result in urination. The dog is then gradually encouraged to walk with only enough support (by grasping the base of the tail) to prevent dragging of the legs. The use of a mechanical cart at this point is contraindicated since the dog tends to rely on it instead of using and developing its own support.

Patience in gaining the dog's confidence is necessary; otherwise, a dog in pain may resent being moved and try to defend himself by biting. During the postsurgical recovery time, usually four days to three weeks, the dog will regain control of urination, defecation, and walking, although complete recovery in the latter may take

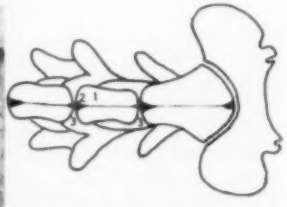

Fig. 11—After the musculature has been bluntly dissected from the vertebrae, the disc is curetted with the needle and tartar scraper; (1) body of vertebra, (2) ventral prominences adjacent to the disc, and (3) disc.

several more weeks. The average hospitalization period is from two to three weeks.

TREATMENT RESULTS

The data on the total number of cases of disc paraplegia seen by the author from 1950 to 1956 has been accumulated and analyzed. Of the 154 cases seen, 46 have been discussed in a previous publication.[10] The breed incidence of the 154 cases was: Dachshund, 61 (40%); Cocker Spaniel, 47 (30%); Pekingese, 11 (7%); Beagle, 11 (7%); and others, 24 (16%). The average age was 5.2 years. The sex distribution was 89 (58%) males and 65 (42%) females.

Of the 154 animals, 80 were treated surgically, 62 conservatively, and 12 were not treated. Those not treated will not be discussed since they were largely hopeless cases, or so mild they needed no therapy. Questionnaires were distributed to all owners of dogs treated more than six months ago. Some were not returned. Of the 68 cases on which reports were returned (47 surgical and 21 conservative), the post-treatment observation time was from six months to four and one-half years. The average observation time for the surgical cases was 1.3 years and for the conservatively treated cases 1.4 years. Four dogs treated surgically were still entirely normal four years after surgery. The overall results of the surgical and conservative treatment categories are shown (table 1).

TABLE 1—Results of Surgical and Conservative Treatment

Treatment	Good	Fair	Poor (noncomplicated)	Poor (complicated)
Surgery 80 cases	53 (45*) 66%	7 (1) 9%	10 (0) 12%	10 (1) 12%
Conservative 62 cases	14 (8) 23%	18 (12) 28%	16 (1) 27%	14 (0) 22%

*Figures in parenthesis are numbers of dogs with more than a sixth-month post-treatment observation time, as reported in questionnaires or seen personally.

DISCUSSION OF RESULTS

The classification terms used in table 1 include: "good," indicating a normal recovery; "fair," indicating a marked improvement but not entirely normal; and "poor-complicated," including those that died or were destroyed primarily because of a concomitant complicating condition, usually a severe cystitis from improper urination, toxemia from improper elimination of feces or urine, or the acute myelitis condition which has been mentioned previously.

It is significant that many dogs were treated surgically after conservative measures had failed. As a result, many were not suited for surgery because of the long duration of the condition. These factors, as well as the so-called "hopeless" cases, which were operated upon at the owner's insistence, make the "good results" figure in table 1 lower than would be expected if all cases had been properly selected. Of the conservatively treated animals, 6 of the 14 in the "poor-complicated" category were considered acute cases (progressive hemorrhagic myelitis usually causes death in a few days in this type).

There has been no evidence of a recurrent protrusion in a surgically treated case in which the disc had been properly curetted. This conclusion is based on a study of many follow-up radiographs and necropsies. Symptoms can recur, postsurgically, in discs which have not been removed. Owners should be advised of this possibility where spinograms show multiple disc degeneration.

Five dogs had other discs affected within one and one-half years after surgery, 3 of these developed cervical protrusions after a lumbar disc had been surgically treated and 2 recovered with treatment (1 surgical). The third dog was destroyed because of the presence of multiple disc protrusions. Two of the 5 dogs were destroyed without a necropsy.

SUMMARY AND CONCLUSIONS

The data pertaining to 154 cases of disc protrusion in the dog have been presented. Of the 154, 68 have been followed up by questionnaires or by personal examinations after six months to four and one-half years (av. 1.4 years). It is concluded that surgical treatment, consisting of a combination of disc fenestration and hemilaminectomy, is a highly satisfactory procedure in suitable cases.

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Meat Hygiene

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The field of meat hygiene belongs to the veterinary profession. Thorough antemortem and postmortem examinations of food animals are basic to the production of clean, sound, wholesome meat. Under the systems of education with which we are familiar, no other profession is qualified through training and experience to perform this service.

A complete meat hygiene program must satisfy all of the needs of the consumer. It must be designed to prevent the transmission of disease from animals to man. However, it must also assure the consumer that the meat came from healthy animals, that it was produced under sanitary conditions, contains no harmful preservatives, is not adulterated, has not been altered or treated to conceal inferiority and, finally, that it is truthfully and informatively labeled.

The Food Hygiene Committee of the AVMA has prepared and distributed, to local and state governments, model legislation for an adequate program. It provides for veterinary supervision for all slaughtering operations. The heads of such programs correspond freely with federal officials when guidance is desired.

Adequate financing is essential to an acceptable program. Two things are particularly important: (1) Salaries must be sufficient to attract and hold an adequate staff and, (2) the finances must be so controlled as to assure no financial transactions between the owner of the animals or the establishment and the inspector. The integrity of the service can be no better than the integrity of the official making the inspections.

The federal government operates only in those plants that prepare products for interstate or foreign sale. Individual states and cities, which usually follow the federal program, are responsible for meat hygiene at the state or local level. In the United States, approximately 80 per cent of all commercially produced meat is prepared under the federal program. During the year ended June 30, 1956, antemortem and postmortem inspections were conducted by federal inspectors on 108,539,755 animals as follows: cattle 19,679,898; calves 7,606,018; sheep 14,208,652; goats 85,736; swine 66,779,920; and horses 179,531.

A well-organized meat hygiene program covers all processing operations, such as curing, smoking, chopping, canning, and rendering, and assures the acceptability of all substances used in the preparation of meat food products that may come in contact with such product or which may contribute to good sanitation. During the past year, we supervised the processing of over 18 billion lb. of meat food products.

A healthy livestock population is assured when there is close cooperation between officials in meat hygiene and in animal disease control. Often, the existence of a disease is first observed by the veterinarian in the meat hygiene program. Prompt diagnosis and control procedures then team together to protect the livestock population. This cooperation is well demonstrated by our experience with bovine tuberculosis which has been reduced to less than 0.1 of 1.0 per cent.

In the United States, all slaughtering operations are under the direct supervision of veterinarians. When additional inspectors are warranted, laymen are employed to assist veterinarians. The lay meat inspectors are trained to detect abnormal or diseased conditions and they have the affected carcasses retained for veterinary

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disposition. They are also used to supervise meat-processing operations.

The disposal of condemned or rejected products deserves careful attention. They must be destroyed in a manner that will make them unavailable for human food. When pathogenic organisms are involved, the disposal should be worked out with disease control officials so as to assure no further dissemination of disease.

Eight laboratories are provided to service inspectors in the fields of chemistry, physics, nucleonics, electronics, microbiology, pathology, parasitology, and serology. Prompt reports from these laboratories to the inspector permit him to make dispositions based on his total knowledge of the problem.

The last 15 years have brought marvelous developments in the use of chemicals, antibiotics, growth-promoting substances, and irradiation. Space does not permit a discussion of what each may contribute to meat hygiene, but it is our responsibility to evaluate all of these developments in order to (1) assure the protection of the welfare of the consumer, and (2) to provide for full use of these developments by the producer.

As members of the veterinary profession, we must continue to strive for more adequate coverage of this essential service.

Importation of canned pork into the United States increased to 113 million lb. in 1954; 107 million lb. in 1955. One-third of the canned hams consumed were imported chiefly because of their higher quality and less fat. Of the 11 billion lb. of pork consumed in 1955, 1.6 per cent was imported. Pork imports are three times that of beef and veal.—*Prairie Farmer, Oct. 6, 1956.*

Water Balance of the Camel.—While other animals die from circulatory failure when their water loss exceeds 12 per cent of their body weight, camels can be dehydrated 30 per cent. Unlike many other mammals, they do not lose their appetite when deprived of water until dehydration is severe. When on a dry ration, their urine output is low (0.5 to 1.0 liter/day), their feces have a low water content, and little water is lost by evaporation.—*Vet. Bull., Sept., 1956.*

Plagues and Contagions in Antiquity

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Epidemics and epizootics played a part in the life of all peoples. The poets sometimes exaggerated them and historians did not fully appreciate their consequences.

Some of them came from germs that were essentially saprophytic and developed in the soil in one or several continents over a period of geological eras. Because the places in which diseases developed did not extend beyond narrow confines, the various peoples throughout history often had to leave the affected areas in order to search for healthier places in which to live.

Through the ages, some epidemics came about as a result of an intimate combination of exceptional circumstances among the animal or human populations. Some diseases definitely disappeared; others reappeared later because the germs were either conserved in the surroundings or in living carriers; while still others finally became permanently established by uninterrupted transmission from one individual to another. This may have occurred either when the disease first struck or after several epidemics.

The frequency and seriousness of the epidemics became greater in proportion as the human and animal groups became significant. However, the dangers remained limited since the isolated populations were exposed only to local infections. With the formation of large groups constituting nations and armies and the intermingling of peoples, a result of wars and migrations, epidemics became more frequent and more dreadful.

Epidemics are reported in the first prehistoric documents and they have continued to become more frequent up to modern times. They caused frightful disturbances in all parts of the world and sometimes decided the existence or destiny of peoples.

The first specific documents concerning epizootics are furnished by the Bible and date from about 1,750 B.C. Of the ten plagues that Jehovah sent the Egyptians, who had enslaved the people of Israel, the

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Translated, edited, and modified from Leclainche, E.: Histoire de la Médecine vétérinaire. Office du Livre, Toulouse (1936): 64-68.

fifth and sixth attacked animals. The fifth plague is summarily mentioned in the Book of Exodus, chapter IX, verse 3. The threat was effectively carried out and "all the animals of Egypt perished." The sixth plague attacked only man and horses (Exodus, chapter IX, verse 9).

In the seventh book of the "Metamorphoses," Ovid described a plague that depopulated the Aegean Isle during the war between Minos and the Athenians in 1,295 B.C. The disease first attacked dogs, birds, cattle, wild animals, livestock, and rural inhabitants, and then spread into the towns.

Homer, in the first book of the "Iliad," (app. 1218 B.C.), spoke of a plague in the camp of the Greeks during the siege of Troy. This time, again, dogs were struck first, then horses and mules, and finally man.

Plutarch said that in the time of Romulus, 753 B.C., there was a general mortality among men and animals: "One died almost as soon as one was stricken."

There was another disease common to men and animals observed by the Latin authors, by Tite-Live in particular, which was described with such insistence and precision that there is no doubt of its existence during the five centuries before the Christian era. Denis of Helicarnasse observed it in 488 B.C. In 461 B.C., the contagion was brought to Rome by the peasants who were forced back to the city with their animals by the Volscian invasion. In 451, 430, 424, and 397 B.C., the plague appeared again at Rome and in the neighboring countryside.

In 212 B.C., the Roman and Carthaginian armies, fighting in Sicily, were decimated by a disease that the poet Silius Italicus precisely described. This time, again, the dogs were the first to be infected, followed by birds, livestock, and man. The symptoms were those of a septicemia with sudden onset, chills, high fever, dehydration of the mucous membranes, and hot breath followed by pulmonary localization with cough and sanguineous nasal discharges, terminating in emaciation and death.

It is the same epidemic that Thucydides described:

> It was a plague that killed a great many men and animals. The dogs were the first to catch it. The weakened birds did not have the strength to hold themselves in the air; the cadavers of the fauna lay in the forests. The horrible plague attacked the armies and death spread everywhere.

It must be considered that besides the human groups whose history is somewhat known to us, others, spread all over the face of the earth, have probably also suffered the attacks of the same human and animal contagions, to which we sometimes get reference from legends or mythology or which have entirely disappeared from man's memory.

One can sense what these catastrophes must have been to primitive humanity which had no protection against the rigors of climate or the whims of reason. In organized society there was no less difficulty. Virgil did not exaggerate when he described the state of affairs in the Nordic countryside after a bovine plague had occurred:

> It was then that in these sad countrysides, one could not find two oxen that were alike so that they could be hitched to a wagon which carried offerings destined to the temple of Juno. Men could be seen reduced to digging up the ground with hoes, burying the seeds with their nails and pulling the heavy wagons to the tops of the hills.

Virgil's picture of the dangers of the contagion, found in "Georgics," Book III, V, contained the essential facts even though they were embellished by a poet:

> The dead animals could not be skinned, nor use made of their skins, because they could not be purified by air or even by fire. Sick sheep could not be shorn, nor could the fleece which [was] impregnated with gnawing poison be removed, nor could they touch its contaminated wool.

Neither the power of the gods nor the knowledge of men could stop the disease. "It had conquered the science of the masters, that of Chiron of Milampus. Escaped from the tomb in the Styx, Paletisiphone lashed her fury. . . ."

The epizootics are often mentioned or described in records of antiquity. Lucretius considered them rather common in his time. He described the "holy fire," *ignis sacer*, which was often mentioned afterward.

In describing the holy fire in the various species, Virgil gave us some precise data that permits us to recognize, almost with certainty, the existence of pleuropneumonia of cattle, typhoid fever of the horse, sheep pox, measles of the pig, and rabies of the dog. Collumella described pleuropneumonia. The *mentigo* or *ostigo* to which the shepherds referred were malignant ulcers of the lips and mouths of lambs and kids and seems to have been a serious form of ecthyma.

Other descriptions do not correspond to any present day diseases. Sheep plague, the *caprarum pestilentia* of Collumella, was neither pox nor anthrax. It decimated or destroyed the herd and the sick sheep were killed to arrest the contagion. Sheep pneumonia, the *coriago* of the peasants of Latium, which was similar to that of pigs and that had to be treated in the same way, does not correspond to any of the present forms.

The plague that Ovid and Homer described still raged in the early centuries of the Christian era. Toward 130 A.D., there was an epidemic that affected all of Italy and, according to Herodian, many men and animals died. In 216, another invasion attacked Italy.

In the epizootic of 376-386, bovine plague was positively identified for the first time. The dialogue poem by Severus Sanctus, the chronicle of Cardinal Baronius, and the one by Ambrosius are valuable documents. The disease, which came from the Orient, devastated Belgium, Flanders, Pannonia, and Illyria before reaching the Roman countryside.

Bovine plague again raged in central and southern Europe during the fifth century. And for many centuries more, suffering humanity has endured, with the same resignation, the attacks of the same diseases.

Bovine Anthrax Traced to 1952 Bone Meal Episode.—Anthrax appeared in July, 1956, in cattle on a pasture in Ohio, on which hogs developed anthrax in 1952, presumably due to being fed contaminated imported bone meal. This is the first known report of anthrax reappearing on fields contaminated from that source.—*Anim. Dis. Trends, Ohio S.V.M.A., July, 1956.*

Oldest Captive Gorilla.—Bamboo, a male gorilla who now is 30 years old and weighs well over 400 lb., was brought from West Africa's tropical forest to the Philadelphia Zoological Garden in August, 1927, when about 1 year old. His age is comparable to that of a man of 60.—*Sci. News Letter, Sept. 15, 1956.*

Tranquilizing drugs composed three of every ten prescriptions being dispensed today in the high frequency category.—*J. Am. Pharm. A., Oct., 1956.*

Age Changes in the Common Integument and Sense Organs of the Dog

Studies have been in progress in the Department of Anatomy, Division of Veterinary Medicine, at Iowa State College for the past three years on age changes in the common integument and sense organs of the dog.

Skin samples from 12 different areas from 13 normal dogs ranging in age from birth to 28 weeks were studied microscopically. Observations were made on the morphology, location of glycogen, and distribution of alkaline phosphotase in normal canine skin at various ages. The hair follicles were observed to develop from a simple form at birth to a compound form at 28 weeks of age. The sudoriparous and sebaceous glands were well developed and functional at birth.

Age changes in the lingual papillae of 31 dogs were studied and the pattern of their structure and distribution was established.

The eyes of 16 dogs were studied macroscopically and microscopically to determine the structure of the mature eye and the growth changes that occurred from birth to 2 years of age. At birth, the eye had the form of a sphere; at the age of 6 to 8 weeks, it had the form of an oblate spheroid, the typical mature form of the mammalian eye. The eye grew most rapidly the first eight weeks of postnatal life and reached growth maturity when the dog was 28 weeks old. Specific details of structure that varied from the human eye were discussed.—*R. Getty et al. in J. Gerontol., Oct., 1956.*—B. FORSTER.

Insects can now be tagged with radioactive chemicals to study their living habits, as well as their movements among animals of one or more species, and their length of life. Fleas have been difficult to tag but now can be dipped in a solution of tenaciously adherent "cerium 144" which can be detected with counters. This method may be useful also in studies of ticks, lice, and other insects.—*Sci. News Letter, Oct. 13, 1956.*

Alcoholism is said to be second only to war as a destroyer of human life.—*Sci. News Letter, Sept. 1, 1956.*

Surgical Conditions Affecting the Reproductive Tract of the Bull

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AMONG THE most perplexing problems veterinarians are called upon to solve are those involving treatment of the bull, particularly the range bull which has never been handled. These animals must be restrained in order to make a careful examination and diagnosis. Many of them are vicious, or become so when they are subjected to conditions to which they are unaccustomed, and will fight at the least provocation. Facilities for handling these animals are often lacking and, therefore, a portable chute is practically indispensable. However, there is still the problem of how to get the bull in the chute.

When a chute is not available, the mature bull can be roped, pulled up to a strong fence post, and haltered, using a ¾-inch rope halter with a long lead. It is often advisable, or even necessary, to administer a dose of chloral hydrate in order to quiet the animal. This can be given either intravenously or by means of a stomach tube. The latter is usually preferred as it is easier and safer to snub the animal to a fence post and pass a tube than it is to give an intravenous injection. In some instances, it is preferable to have drinking water withheld and then have the owner add the chloral hydrate to it and notify the veterinarian as soon as the bull drinks. This method does not work well with Santa Gertrudis or Brahman bulls as they are naturally somewhat shy. If the bull must be handled after surgery, a bull nose ring should be inserted.

The most important affections of the bull which require surgery are those which involve the reproductive tract. A few of the most common ones are described.

HEMATOMA OF THE PENIS

This condition is generally caused by the penis being sharply bent, rupturing the corpus cavernosum during breeding. The area of injury, usually just anterior to the scrotum, is manifested by a firm swelling which may extend to the end of the sheath, even pushing the prepuce out of the orifice. It may be difficult to differentiate between this condition and a hematoma in other tissues in this region, except that the enlargement is generally freely movable under the skin.

An operation is imperative if the bull is to be returned to service. However, we usually wait a week or ten days to allow some of the swelling to subside and to allow a good, firm clot to form. Food is then withheld for 24 hours, after which the bull is usually given a narcotizing dose of chloral and is restrained in a lateral "stretched out" position. After the operative area is prepared and anesthetized with a 2 per cent procaine solution, an incision is made in a downward and forward direction over the most prominent part of the swelling until the tunica albuginea is reached. The latter is then similarly incised, the firm blood clot is completely removed, and the tunica albuginea is closed with several interrupted sutures of chromic catgut, leaving a small opening for drainage. The skin incision is then partly closed with interrupted sutures of noncapillary braided silk, again leaving an opening for drainage. A gauze seton saturated with B.I.P. is inserted through the openings and is left in the cavity for 48 hours. It is replaced as often as seems necessary. Three or four weeks is required for healing and about three months for the bull to be ready for service.

HEMATOMA OF THE PRESCROTAL TISSUES

Hematomas involving the tissues just anterior to the scrotum, with no involvement of the penis, occur frequently. These hematomas usually have a history of having been present for some time and frequently terminate in abscesses. Aspiration will determine whether the enlargement contains pus or blood. In either case, the procedure is essentially the same, but a hematoma must not be opened too early.

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The bull should be anesthetized and restrained, as in the previous condition, with the most prominent side of the enlargement uppermost. However, both sides should be prepared for surgery. Evidently these hematomas are due to some severe contusion for in most instances the structures beneath the skin are severely lacerated. After the swelling has been incised and evacuated, digital palpation should determine if and where an incision should be made on the opposite side. This is generally necessary as the penis causes a median barrier preventing drainage from one side. After the hemorrhage has been controlled, a seton saturated with B.I.P. is drawn through the openings and is tied in place. This seton may be changed as often as is necessary, but should be used until the cavity of the abscess or hematoma is practically filled in. Most of these bulls go back in service in about three months.

OTHER INJURIES TO THE PENIS OR PREPUCE

Bulls, in some manner, frequently lacerate the distal portion of the penis or prepuce, or both. Considerable swelling and phlegmon occur and adhesions of the prepuce readily develop. Unless these adhesions are broken down, the bull is unable to protrude the penis and should be sent to market. Most of these cases can be corrected if the owner will accept the expense of treatment. Treatment must be employed until the acute inflammation subsides, which requires several days. Hot applications applied daily are excellent.

Prior to surgery, the bull is given 10 to 15 cc. of a 2 per cent solution of procaine epidurally. (If too much is given, he is unable to retract the penis and damage may occur when he attempts to get up.) He is then cast and hog-tied; the two front feet and the lower hindfoot are tied together. A rope is then put on the upper hindfoot just above the fetlock, carried forward over the withers, and around under the neck, pulling the leg forward and upward. The free end of the rope is then tied just above the hock using a double half-hitch. If difficulty is encountered in securing the penis, a few whiffs of chloroform will facilitate the procedure. The end of the penis is grasped by an assistant, using a small towel. The operator then pulls it out, carefully breaking down the adhesions by tension, manipulation and, in some instances, with scissors. The tissues may tear, requiring a few interrupted sutures with chromic catgut. The penis is then smeared with B.I.P. and pushed into the sheath. If it will not remain retracted, a suture may be placed in the end of the sheath and removed later.

The above procedure must be repeated once or twice a week and then less often until adhesions no longer form. After the initial treatment, the bull can often be secured in a chute and rectal massage applied until the penis can be grasped and treated. Many valuable bulls can be saved by the above procedure.

Congenital Adhesions of the Prepuce and Penis in Young Bulls.—Occasionally, a young bull can not protrude his penis completely, it being bent ventrally due to cordlike adhesions of the penis to the prepuce. We recently encountered a young Charolais bull in which there were three such congenital adhesions.

Surgery is indicated. The penis is easily protruded and the cords are excised at their attachment to the penis and to the prepuce. The hemorrhage is negligible and, generally, no aftercare is required.

Prolapse of the Prepuce and Circumcision in the Brahman and Santa Gertrudis.—Prolapse of the prepuce is a complication often seen in Brahman and Santa Gertrudis bulls (fig. 1). These bulls have an enormous pendant sheath, and the prepuce is extensive. It often prolapses and is easily traumatized by various plants or objects and, eventually, edema, fibrosis, and an enlargement of varying size develop. Removal of the enlargement by surgery is the only means of returning these bulls to service. An operation should not be attempted

Fig. 1—Prolapse of the prepuce of a Santa Gertrudis bull.

until the edema has receded and the area is soft and pliable.

Restraint is a problem since most of these animals are easily excited. Even those that are reasonably gentle do not like strangers. If a good strong chute is not available, they will have to be pulled up to a strong fence post and haltered. With the fence between the operator and the bull, a stomach tube is passed through the nostril and 2 to 4 oz. of chloral hydrate is administered. About 20 to 30 minutes later, the bull is cast and restrained in a lateral, stretched out position, preferably on the right side, in case tympany should develop. The parts are thoroughly scrubbed, shaved, and anesthetized locally with 2 per cent procaine. The prepuce is pulled through a hole in a large sterile shroud in preparation for amputation. The external layer of the prepuce is incised transversely just below where it joins the skin and is stripped down slightly. The internal layer is then partly incised and the two layers are sutured together with interrupted sutures of noncapillary braided silk. If not sutured as it is incised, the loose end may retract out of reach. Hemorrhage should be thoroughly controlled. To eliminate dead spaces, the fascia should be drawn together in the wound. This also lessens the size of the wound, facilitates suturing the prepuce, and helps to eliminate the edema and swelling which often occur several days after the operation. To avoid a stricture, a triangular area of skin, about 3 inches long, may be removed ventrally at the prepucial orifice. The prepuce is then slit medially and sutured to the skin on its respective side.[1] The bull is released and put into a clean pen after B.I.P. is applied to the wound. Little aftercare is necessary.

[1]Frank, E. R.: Veterinary Surgery Notes. Burgess Publishing Co., Minneapolis, Minn. (1947): 234.

In an attempt to reduce dystocias in heifers, cattlemen have often bred them to Aberdeen Angus bulls expecting the calves to be smaller. In one test with Hereford cows, the calves sired by Angus bulls averaged only 1 lb. less than those sired by Hereford bulls. The bull calves averaged 64 lb. as compared with 65 lb. for purebred Herefords; the heifers 58 lb. compared with 59 lb. The age of the sires made no difference in the size of the calves.—*Farm J., June, 1956.*

Anal Suture for a Prolapsed Rectum

Prolapse of the rectum, which occurred shortly after farrowing, was successfully corrected in 23 sows by placing a steel wire suture (No. 32) in the tissues deep to the internal anal sphincter so that the anus was circumscribed about ½ inch from its border. An eyeless, curved needle with the wire attached was used. The prolapse was then reduced and the wire tightened so the anus would admit two fingers. The knot was buried in the tissues, and the suture was left *in situ.*

In 4 small pigs, 20-day chromic catgut was similarly used with excellent results.—*R. Richardson in Vet. Rec., Aug. 11, 1956.*

Effect of Starvation on Sperm Count.—Certain wild animals (salmon, goose, fur seal, penguin) fast during the mating season. Furthermore, plants raised in inadequate soil produce more seed than those grown in fertilized soil.

When male dogs were starved for five or ten days, the sperm count of 3 of 5 increased during starvation while the animals were losing from 3 to 8 lb. of weight, and then returned to normal when the dogs were again fed.—*Science, Aug. 24, 1956.*

Persistent Hymen in a Mare.—A gray Hunter mare, 11 years old, became vicious each time an attempt was made to breed her. A persistent hymen "somewhat similar to that found in the typical white heifer disease of cattle" was found in the vagina. It was thin and flexible, with a central opening admitting three fingers. Four months after this membrane was removed, by four radiating incisions, the mare was bred and conceived.—*A. S. Harris in Vet. Rec., Sept. 15, 1956.*

Antioxidant (DPPD) *Inhibits Parturition.*—A feed grade of diphenyl-*p*-phenylenediamine, at levels only several times in excess of those fed to chicks to prevent encephalomalacia, induced prolonged gestation in rats and, at parturition, a high mortality both in the mothers and the overdeveloped fetuses.—*J. Agric. and Food Chem., Sept., 1956.*

An Angus cow in Illinois gave birth to a dark-colored calf in June and then, 5 weeks later, to another calf, this one black and white.—*Prairie Farmer, Sept. 15, 1956.*

Rupture of the Prepubic Tendon in a Cow

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Rupture of the prepubic tendon in the cow is considered a rare condition[1]; therefore, this case is reported.

A crossbred cow, 5 years old, had been kept as a nurse cow in a herd of Hereford cattle. She had twice calved unassisted and was expected to have another calf during the week in which the symptoms developed. For four days, she had shown a gradual increase in abdominal girth; the owner stated that the cow had "dropped in the belly." There was no history of trauma and the cow had been eating and defecating normally.

Examination on April 22, 1956, revealed extensive edema of the posterior abdominal wall and a greatly distended abdomen (fig. 1). The abdomen was definitely "pear" shaped when viewed from the rear (fig. 1). The pressure and edema had impaired circulation to the udder and teats, resulting in necrosis of the teats. Examination, *per vagina*, revealed a closed cervix drawn considerably downward and forward. Her temperature was normal and she could rise and walk unassisted. The alternates of euthanasia or cesarotomy were offered and the latter was chosen.

The surgical procedure, the next morning, was as follows. A chute was employed for restraint in the standing position. The left flank was clipped and scrubbed with a solution of quaternary ammonium chloride compound. Anesthesia was accomplished with 3 per cent procaine solution infiltration of the proposed line of incision; 10 cc. was injected at 3-inch intervals. An incision, about 24 inches long, was made in the paralumbar fossa, posterior to last rib (fig. 2). Upon penetration of the peritoneal cavity, a colorless watery fluid was encountered, perhaps 30 gal. of which escaped.

The uterus was exposed by manipulating the rumen. Four large loops of umbilical tape were placed in the wall of the uterus and it was pulled up to and out of the abdominal incision. When the uterus was incised, another large quantity of watery fluid escaped. A normal calf was delivered and the fetal membrane was removed with ease. The uterus was closed using a double row of Lembert sutures with No. 2 chromic gut.

A sterilized stomach tube was used to siphon several more gallons of fluid from the peritoneal cavity. The abdominal wall was closed with interrupted figure-8 umbilical tape sutures placed at 2-inch intervals. This suture penetrates the skin, crosses the wound, penetrates the muscle and peritoneum, and then is reversed through the opposite structures.

Fig. 1—Right lateral view (left) and posterior view (right) of the cow before the operation.

Supportive therapy included 500 cc. of 50 per cent dextrose and 500 cc. of calcium gluconate solution intravenously; 30 mg. of stilbestrol, 3,000,000 units of procaine penicillin, and 5 Gm. of dihydrostreptomycin intramuscularly; and 250 cc. of sulfamethazine solution intraperitoneally.

The cow ate, drank, and appeared bright, but was found dead on the third postoperative day. A necropsy was not made.

DISCUSSION

The cause of this condition was not evident. Whether the prepubic tendon rup-

Fig. 3—Left view of a cow; postoperative, showing sutured incision.

Dr. Whalen is a general practitioner in La Salle, Ill.

[1]Williams, W. L.: Veterinary Obstetrics. Published by the author, Ithaca, N. Y. (1948): 288-294.

tured because of the weight of the fluid, or whether the fluid accumulated as a result of the impaired circulation, could not be determined. Since the fluid was colorless, there would seem to have been little, if any, hemorrhage. The condition could have started as hydrops amnii, but this seems doubtful since the fetal membranes were not edematous and the calf was normal.

The prepubic tendon was totally separated from the pubis. The stretching had torn the peritoneum in the prepubic region into many (perhaps 20) longitudinal strips an inch, or more, wide and with rents up to 10 inches long.

The cause of death is speculative. From the owner's report it seemed more like shock than peritonitis.

Notes on Cesarotomies in Cows

In 18 months, to March, 1956, cesarotomies were performed on 54 cows at the University of Bristol, England. All were sent to the hospital by practicing veterinarians. The indications for the operation were 11 monstrosities, 8 oversized fetuses, 11 undilated cervices, 8 malpresentations, 6 torsions of the uterus, 2 fractured maternal pelves, 2 hydrops amnii, 2 mummified fetuses, and 4 miscellaneous.

Of the 54, 74 per cent were dairy cows, although only 48 per cent of the cattle in the region were estimated to be of dairy breeds. All but 3 were operated on, while standing, through a vertical incision about 14 inches long in the left flank, using paravertebral anesthesia. The advantages of this method are the reduction of shock by less handling of the small intestines, no interference from the omentum, flank wounds are more easily sutured and heal better than ventral incisions, and asepsis is more easily maintained. However, if the cow falls, difficulties may arise.

The incision, over the fetus, in the pregnant horn should be parallel to the long axis and as far forward as possible to facilitate suturing. The uterus is repaired with a No. 4 catgut continuous Lembert suture, inverting 1 to 1½ inches of the edge of the uterine wall. The rapid involution makes it advisable to start the sutures at the posterior extremity of the incision and to draw them tight. Fluids which escaped into the peritoneal cavity should then be aspirated and the external wound closed.

The fetal membranes were removed during the operation or were expelled within 24 hours in 75 per cent of the cows; in 1 cow, they were retained 11 days with resulting metritis. Histories subsequently obtained on 29 of the 39 survivors showed that 8 had again calved, 5 were pregnant, and 16 failed to conceive.—*Vet. Rec., Aug. 25, 1956.*

Reproductive Potential in Chinchillas.—Although virgin mature chinchillas averaged five primary corpora lutea and pregnant females 4 per estrus, the national average for a litter in 1953 was 1.05. The average number of young per female per year was 2.1 born and 1.68 surviving at 30 days. In contrast, laboratory rats usually ovulate 10 eggs and produce 9 young per litter, and the spiny hedgehog of Madagascar has up to 24 young per litter; 1 had 32, the largest litter ever reported for a mammal. The Virginia opossum and an insectivore (Elephantulus) may produce 100 or more ova per estrus but only 1 or 2 are implanted.—*NCBA Res. Bull., May, 1956.*

Rate of Sperm Transport in Female Rabbits.—Using giant white does, bred once, laparotomies were performed at various intervals postcoitum, fallopian tubes were double ligated and sectioned, and two days later the does were killed and the tubes flushed to recover the ova, which were then examined for evidence of fertilization. Sufficient sperm were present in five hours, in all cases, to fertilize all the ova. Differences in the uterine motility and in the mechanical barriers offered by the cervix and by the tubal junction probably account for the variability in sperm progress.—*Science, Sept. 28, 1956.*

Embryos developed from the one undestroyed cell of the four-cell stage of fertilized rabbit ova. (After each of the first two cell divisions, one of the two cells had been destroyed by being punctured.) When these treated ova were fertilized and implanted in a uterus for seven to nine days, embryos developed in 18 of the 30 trials. Some of the resulting embryos were complete and symmetrical, others were blastocysts with no organized embryonic development.—*Science, Aug. 31, 1956.*

The gestation period is longer for males in cattle, horses, and possibly sheep and camels; for females, in man and possibly in guinea pigs.—*Vet. Bull., Sept., 1956.*

Experimental Vibriosis in Sheep

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VIBRIOSIS has long been recognized as a disease of cattle and sheep. With the concerted efforts toward the elimination of brucellosis, *Vibrio fetus* has rapidly become recognized as one of the major causes of reproductive failures in cattle. The sheepmen of the West have long been harassed by abortion storms caused by *V. fetus*.

In sheep, much is known about the epizoology of the disease but very little is known about the natural reservoirs of infection, natural means of transmission, and pathogenicity of the organism.

The purpose of this paper is to present evidence on a means of transmission which could be natural and to show the period of gestation in which the ewe is most easily infected.

Field observations[4,8,9] and controlled experiments[1,2] have cast doubt on the ram as a serious source for the spread of vibriosis. During the same season as the experiment herein reported, Colorado workers[2] used "clean" rams (that is rams that were from flocks with no previous abortions and from which *V. fetus* could not be isolated) on two lots of clean ewes. The ewes of one lot served as controls and suffered no loss from abortion. The other lot of ewes was fed infective tissues orally during the fifth month of pregnancy and suffered heavy losses from abortion due to *V. fetus* infection. A third lot of clean ewes was bred to an infected ram and suffered no abortion loss. Carrier ewes may be reservoirs for *V. fetus*.[6] The organism has been found in hay, soil, and manure after experimentally contaminating these materials.[3] Recently, researchers at the Wyoming Experiment Station[7] have found many Vibrio-like organisms in known infected ovine fetuses after they were buried in manure for 90 days.

Montana workers[5] and others[5] have reported the routine use of intracardial, intraperitoneal, and intravenous routes for injection of the Vibrio organism into pregnant guinea pigs and sheep to check for pathogenicity of the organism. This appears to be a drastic means of challenge, especially since it is unlikely that natural infection would occur by any of these routes. Many workers have reported carrying pathogenic strains of *V. fetus* in the laboratory. Wyoming workers have been able to carry isolated organisms in pure culture but have been unable to demonstrate them to be pathogenic for sheep after several transfers.

MATERIALS AND METHODS

Fifty virgin yearling ewes were purchased from a band with a history of no previous abortions. They were bred to rams that had never been exposed to vibriosis. The ewes were separated into four lots according to breeding dates.

In lot 1 (12 ewes) were the uninoculated controls. Each of 13 ewes in lot 2 was given 11 inoculations between Feb., 18 and March 2, 1955, with a composite of four *V. fetus* strains (1 bovine and 3 ovine)[a] which were considered to be pathogenic at the time of isolation. The stage of gestation at initial inoculation was 75 to 78 days. Lot 3 (13 ewes) received, on consecutive days, three inoculations of V. fetus from a recent culture of ovine origin. The stage of gestation was 92 to 97 days. Lot 4 (12 ewes) was inoculated twice with isolates from lot 3. The stage of gestation at initial inoculation was 124 to 127 days.

All inoculations were made orally by spreading a medium containing the organisms over chopped hay. No attempt was made to standardize the amount of inoculum. All lambs of all lots, both dead and alive, were cultured for the presence of *V. fetus*.

RESULTS AND DISCUSSION

The results of this experiment are shown (table 1).

From past histories of and experiments with vibrionic abortion in sheep, abortions were expected to occur in infected ewes any time after about 60 days of gestation. It has been suggested[8] that infection with *V.*

From the Department of Veterinary Science and Bacteriology and published with the approval of the director, Wyoming Agricultural Experiment Station, as journal paper No. 86.

The data in this paper were obtained as part of Regional Research Project W-27 (vibriosis).

[a]Ovine strains 2221, 9431, and 22965 were from isolations made by Dr. J. F. Ryff, Wyoming State Veterinary Laboratory, Laramie. Bovine strain P-1 was from Dr. A. Batlin, University of Wisconsin, Madison, who obtained it as strain "Iongo" from Dr. W. N. Plastridge, Department of Animal Diseases, Storrs Agriculture Experiment Station, Storrs, Conn.

TABLE I—Results of Oral Inoculation of Pregnant Ewes with Vibrio Organisms

Lot	No. ewes	Dates bred	Inoculations* Dates	Inoculations* No.	Stage of gestation (days)	Lambed or aborted Dates	Days past inoculation	No. of lambs Total	Alive	Dead	V. Fetus Isolation Alive	V. Fetus Isolation Dead	Remarks
1	12	Dec. 6-15	....	....	.....	April 31-May 17	....	13	12	1	0	0	1 dead probably suffocation.
2	13	Dec. 8-11	Feb. 18-Mar. 2	11	75-78	May 3-9	78-84	8	8	0	0	..	.....
3	13	Dec. 13-18	Mar. 19-21	3	92-97	Mar. 27	7	....	..	4	..	4	.....
						Mar. 29	9	....	..	3	..	3	.....
						Mar. 31	11	....	..	1	..	1	.....
						April 8	19	....	..	1	..	1	.....
						April 19	30	....	..	1	..	1	.....
						April 21	32	....	..	1	..	1	.....
						May 5	46	....	..	1	..	0	.....
						May 16	57	13	1	..	0	..	.....
4	12	Dec. 18-21	Apr. 23-24	2	124-127	May 3	9	....	..	2	..	2	Twins, 1 liver lesion.
						May 5	11	....	1	1	1	1	1 live, very weak.
						May 6	12	....	1	..	1	..	Liver lesions.
						May 7	13	....	1	1	0	1	1 dead, liver lesions.
						May 8	14	....	..	2	..	0	.....
						May 9	15	....	..	1	..	0	.....
						May 16	24	11	1	..	0	..	.....

*Lot 2 inoculated with a composite of 4 organisms (originally thought to be pathogenic) that had been carried for some time (3 bovine and 1 ovine). Lot 3 inoculated with organisms from recent isolates of April, 1955, outbreak. Lot 4 inoculated with organisms isolated from aborted lambs of lot 3.

fetus by intraperitoneal injection can be expected any time during the last 90 days of pregnancy when ewes are inoculated with pathogenic strains of *V. fetus*.

Ewes used in this study were shown to be free of previous infection in that 12 ewes of lot 1 (controls) had 13 lambs. One lamb from lot 1 was found dead. It appeared to be full-term and was culturally negative for *V. fetus*.

Eight normal, full-term lambs were born to 8 ewes of lot 2. All were negative for *V. fetus*. This lot of 13 ewes, by chance in misbreeding and grouping, had 5 unbred ewes.

Since no abortions occurred from the composite of organisms used in lot 2 and a current culture (ovine) became available, it was used for the inoculation of ewes in lot 3. Eight of 13 ewes in lot 3 aborted within 14 days after inoculation. The latest fetus from which *V. fetus* was recovered was aborted 32 days following inoculation.

The organism used to inoculate ewes of lot 4 was cultured from an aborted fetus of lot 3. *Vibrio fetus* was cultured from aborted lambs as well as from some of those born alive in lot 4 (table 1). All except 1 of the lambs in lot 4 were aborted or born within 15 days after inoculation. *Vibrio fetus* was recovered from 6 of the first 7 lambs aborted and from none of the last 4. All of the observed liver lesions, sometimes described as being characteristic of *V. fetus* infection in sheep, were in lot 4 or from those animals closest to term when infected.

These findings suggest that when 1 ewe aborts in a band, regardless of the source of her infection, the susceptible animals of the entire band are endangered.

SUMMARY

Evidence is presented to indicate (1) that oral inoculation of susceptible ewes is a satisfactory route of challenge with *Vibrio fetus*, and (2) that the bred yearling ewe, in late pregnancy, is a satisfactory animal for use in testing the pathogenicity of *V. fetus*.

The incubation period of *V. fetus* was approximately two to three weeks.

Exposure of susceptible ewes to pathogenic strains of *V. fetus* at any time after 90 days of gestation may be expected to produce abortion.

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A Report of *Strongylus Asini* from the United States

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Chicago, Illinois, and Beltsville, Maryland

Strongylus asini was originally described,[2] in 1920, in Africa, as a new nematode parasite of the donkey (*Equus asinus*). Specimens were recovered from the cecum and from a liver cyst. Since that initial report, several workers[1,3-8] have reported finding this species in the zebra.

A male Grevy zebra (*Equus grevyi*, Oustalet), between 2 and 3 years old, when it became a member of the Chicago Zoological Park in August, 1954, was found dead on Dec. 18, 1955. Routine fecal analyses during the summer of 1955 had revealed no heavy nematode infection, but extensive liver damage caused by parasitic cysts was found at necropsy. The cysts (fig. 1) were located singly or in groups throughout the liver, which was black and congested. Several coiled worms, which were identified as *S. asini*, were observed between the capsule of the liver and the parenchyma. Death was attributed to peritonitis initiated by the worms, plus extensive liver damage. Some thrombosis of the anterior mesenteric artery was observed. Although the intestinal tract was not examined closely, it appeared normal and no worms were found.

Sectioned and stained cyst material (fig. 2) revealed much fibrous connective tissue, with areas of extensive leukocytic invasion.

Dr. Jaskoski is with the Chicago Zoological Park, Brookfield, Ill., and is assistant professor, Department of Biological Science, Loyola University, Chicago, Ill.; Mr. Colglazier is with the Parasite Treatment Section, Animal Disease and Parasite Research Branch, Agricultural Research Service, U. S. Department of Agriculture, Beltsville, Md.

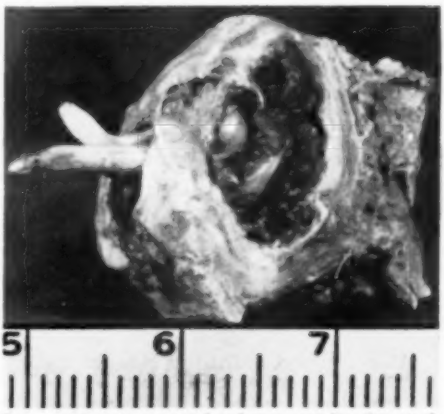

Fig. 1—Hepatic cyst of zebra, partially dissected, showing mature Strongylus asini in situ.

The occurrence of fully developed strongyles in the liver is a point of interest. Immature specimens of *Strongylus equinus* have been found in the liver of Equidae but are more common in the pancreas.

This is the first report of *S. asini* recovered at necropsy of an equine animal in the United States. Thus, it is an addi-

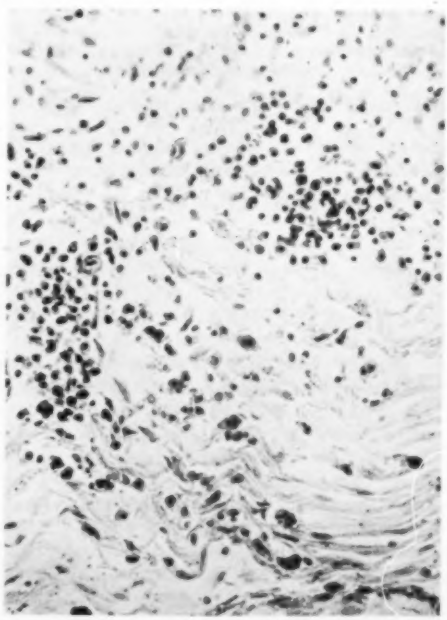

Fig. 2—Section of cyst material from a Zebra, showing connective tissue and leukocytic infiltration. x 288.

tional rarely encountered species that could presumably become parasitic in horses.

Specimens of *S. asini* recovered from the zebra have been deposited in the U. S. National Museum Helminthological Collection (No. 38038) at Beltsville, Md.

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Report on Five Cases of Neurosis in Cows

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Five cases of a neurosis in cows with a common history of having calved within the last two or three weeks are reported. The symptoms varied but all of the cows were extremely nervous.

One cow became entangled in a fence that she knew was there and had respected for a number of years. Her legs, body, and udder were cut severely. There was incoordination, apparent blindness, abdominal pain, and a temperature of 103 F. She was mentally disturbed but not comatose until near the end. Within a few hours she was unable to rise. Treatment consisted of calcium gluconate and dextrose and two small doses of cortisone. She died on the seventh day.

Dr. Kingman, formerly veterinarian for the Wyoming Hereford Ranch, now lives in Mineral Bluff, Ga.

Another cow was staggering, had a wild expression, slobbered profusely, and passed a moderate amount of clear urine (I had nothing but my hat to catch it in, so the urine test was omitted). No unusual odor of the urine, milk, or breath could be detected. The cow's frantic condition made it impossible to give medication intravenously, so 500 cc. of 50 per cent dextrose was given intraperitoneally. While other medication was being prepared, she became quiet and began to eat grass. She was given ½ oz. of chloral hydrate and was turned out to pasture. More of this drug was left for the owner to give but he found it unnecessary.

The condition of the third and fourth cows was much the same as the second except that they were a little slower in recovering, but both were much improved within an hour and required no treatment except dextrose and chloral hydrate.

The fifth cow was violent. She foamed at the mouth and her eyes had a wild expression; the pupils were dilated. Her neck was so stiff that the head could not be lifted more than a foot from the ground. We were too busy to catch a urine specimen. She chewed her legs and sides continuously and was on her knees part of the time. It seemed reasonably certain that we were dealing with "mad itch" or possibly rabies. Both guesses were wrong. She was given 500 cc. of dextrose intraperitoneally and 1 oz. of chloral hydrate orally, cautiously, and with difficulty. The slobbering and "champing" soon subsided and within an hour she was resting but was weak. Another 500 cc. of dextrose (50%) was given intraperitoneally and chloral hydrate was left for the owner to give. The next day we were surprised to find the cow calm and in good condition. No more medicine had been given. Within about four days, the owner reported a relapse; she was acting much the same as before. He was supplied with chloral hydrate and asked to report in case she did not respond. Nothing was reported.

I have hesitated to report these cases since they may be of common occurrence, also because more should have been done toward making a diagnosis. The possibility of lead poisoning, rabies, Aujeszky's disease, and grass tetany were considered. Fortunately, all but 1 cow recovered without a definite diagnosis. The cases were startling and challenging. I received benefit from the experience and from the reading

that it forced me to do. Maybe a simple diagnosis of acetonemia would have saved all that trouble.

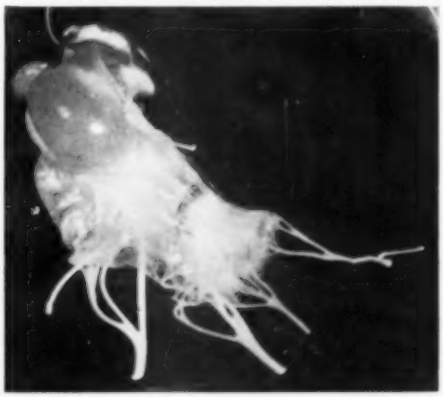
Equine Tetany—Three Cases

Dr. Kingman's report on neurosis in cows brings to mind three cases of equine tetany seen on farms many years ago. On Aug. 8, 1932, a boy's Shetland pony gelding suddenly developed alarming symptoms after drinking water. It frothed at the mouth, sweat profusely, stood rigidly with spasms of trembling, and refused to move. The temperature and pulse were normal. Since the nictitating membrane was relaxed, a diagnosis of tetanus was eliminated. There was no possibility of it having acquired any strychnine or other known poison, so a diagnosis of tetany was made. The pony was given a mild sedative and 125 cc. of calcium chloride solution (20%) intravenously. It was normal in about an hour.

On July 18, 1936, a young draft gelding became restless after working several hours the first day after being on pasture for three weeks. He was unable to open his mouth or swallow, the nostrils were flared and tense, the chin retracted, the masseter muscles quivering, and he walked stiffly and unsteadily. The nictitating membrane was relaxed and the hocks were not abducted, so a diagnosis of tetany was made. He was given 250 cc. of dextrose (30%) and 125 cc. of calcium chloride (20%) intravenously and soon was sufficiently relaxed to urinate, nibble grass, and walk normally. He seemed completely normal 30 minutes after treatment. (One month later, the gelding developed tetanus and died in spite of extensive therapy.)

On Aug. 19, 1939, a draft mare, 8 years old, which had worked only a few hours after being at pasture, began to show stiffness and was turned into a barn lot. A few hours later, the mare was in great distress. Her jaws were set, lips pursed, limbs rigid, and she was sweating profusely. Her temperature was 103 F. She could not be prevented from pressing forward with a stiff, jerky gait. A needle could be kept in the jugular vein only with difficulty, so she was led in a small circle and given a 40-cc. syringe full of calcium gluconate (20%) intramuscularly every time she made a round (total 300 cc.). It was then possible to give 500 cc. of dextrose solution intravenously. In two and one-half hours she was resting quietly and her mouth could be opened about an inch. In another few hours, she was normal.—W.A.A.

A Bulla-like Endocardial Lesion in a Cow



The septal cusp of the tricuspid valve (½ normal size) found at slaughter of an otherwise normal Holstein-Freisian cow, 8 years old, showing a naturally occurring bulla-like lesion on its auricular surface. The dome was 4.0 by 2.5 cm. and 2.0 cm. high. It was 4.0 cm. from the border of the valve.

The endocardium was raised like an epithelial blister. The cause was not evident and extensive inquiry and search of veterinary and medical literature uncovered no report on a similar lesion in any mammal.—Submitted by Drs. Louis Leibovitz, V.M.D., and Albert Schatz, Ph.D., National Agricultural College, Doylestown, Pa.

Intestinal Emphysema in Pigs.—Intestinal emphysema occurs in pigs, sometimes in man, and 1 case has been recorded each in sheep and poultry. All layers of the jejunum and ileum may be affected but the blister-like lesions, from 1 to 20 ml. in diameter, occur chiefly under the serous and mucous membranes. Three cases have been found in several thousand pigs in Poland; 2 were unthrifty but 1 was a healthy pig at an abattoir. The lesions may occur in clusters the size of a fist.—*Vet. Bull., Sept., 1956.*

Dogs as Hosts of Tularemia.—Of 136 dogs tested in Arkansas for tularemia by the serum-agglutination method, 44 were positive in dilutions of 1:20 to 1:2,560. Raccoons, opossums, and possibly cattle also gave positive reactions but, unlike dogs, they do not harbor large numbers of the vector ticks. All of 15 rabbits tested were negative.—*Vet. Bull., Sept., 1956.*

Preliminary Studies on a Canine Respiratory Disease

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THE RESPIRATORY diseases of the dog are of interest to human as well as veterinary medicine, especially since recent work has indicated that the viruses of the canine distemper complex may be etiological factors of disease in man. Additional work may indicate a closer relationship of other canine viruses to human disease. In addition, diseases of the dog create a great problem in laboratory research, since these animals are frequently used as experimental subjects.

In the course of work in this laboratory, many dogs were found to be unfit for experimental purposes due to a chronic anterior respiratory disease. The syndrome of this disease is not typical of any form of canine distemper but, rather, more closely resembles the condition known as tracheobronchitis or "kennel cough." No particular age susceptibility is obvious, although young dogs, in general, show more severe symptoms.

The onset of the disease is insidious, as affected animals show an occasional cough, usually only when excited or forced into action. Coughing is easily induced. There is no obvious loss of appetite and the animal's general physical condition appears to be good. As the disease progresses, spasms of the characteristic dry, "hacking" cough occur more frequently. Often, the coughing raises a small amount of foamy, semipurulent material from the larynx. Even after a particularly severe coughing session, the dog eats and behaves normally.

On auscultation, there are usually no sounds in the thorax, but tracheal râles can be detected in a few cases. A mild conjunctivitis appears in a few animals during the early stages of the disease, but neither the severe purulent eye involvement of classical distemper nor keratitis is observed. After one to several weeks of frequent coughing seizures, the disease gradually subsides, leaving no visible sequelae, such as the encephalitis described for the condition known as "housedog disease" or pharyngolaryngeal tracheitis.[1]

Treatments of various types have been used, including antiserums (distemper and hepatitis) and antibiotics, none of which were beneficial. The symptoms can be partially alleviated, however, with expectorants and mild sedation. Attempts to isolate animals within the kennels by separating cages were not adequate as control measures.

Bacteriological examinations were consistently negative, as were cultures for fungi and PPLO organisms. The condition could not be attributed to any known parasite; therefore, there is a possibility that it may be caused by a virus.

MATERIALS AND METHODS

Animals.—Puppies from mongrel breedings, born and raised in the laboratory, were observed closely for spontaneous disease until 6 to 10 weeks of age, when they were used for experimentation. Only dogs that remained healthy during this entire period were used.

Swiss mice were obtained commercially at 3 weeks of age, except for baby mice which were raised in the laboratory. In most cases, males were used for infectivity trials.

Inoculums.—Original isolates were made from throat washings of naturally affected dogs. The sick animals were anesthetized with nembutal®* and the nasopharynx and tonsillar crypts were flushed with broth which was then re-collected in a syringe. Penicillin and streptomycin were added to these specimens as an aid in the control of bacterial growth, and the broth suspensions were then used for intranasal inoculations of mice.

In mouse passages, lungs and tracheas were removed, homogenated in a Waring-type blender, diluted in buffered (7.2) saline to a 20 per cent suspension, and were treated with penicillin and streptomycin. Mice were anesthetized with ether, and 2 drops of the test suspension were placed in each nostril.

Inoculums that were to be stored were sealed in glass ampules, quick-frozen in a bath of dry ice and alcohol, and placed in a dry ice chest. If material was to be used within 24 hours, it was kept in a refrigerator at approximately 5 C.

Chicken Embryos.—Ten- to 12-day embryonating hen eggs were used to determine whether an infectious agent could be propagated in this medium. Although allantoic injections were used mostly, other routes were tried in a few instances. Five days after injection, the membranes and embryos were examined for gross changes and the fluids

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*Trademark, Abbott Laboratories, North Chicago, Ill.

were tested for infectivity and the presence of hemagglutinins.

EXPERIMENTAL PROCEDURE

The contagious nature of the disease was demonstrated repeatedly by placing healthy dogs in direct contact with affected ones. Susceptibility of puppies was further demonstrated by inoculating 3 intratracheally with 0.5 ml. of a 20 per cent suspension of lung and trachea of an infected dog. Six days later, all 3 puppies showed a nasal discharge and a low, harsh cough. After about one week, they recovered without therapy. The results were negative when 3 litter mate controls were similarly treated, using sterile saline as an inoculum.

Mouse passages were made, using dog throat washings (table 1). Beginning with the fifth serial passage, some of each group of mice showed changes indicative of infection. Lesions, confined to the respiratory tract, consisted of consolidated patches in one or both lungs; the condition was histologically classified as a bronchopneumonia. These animals were not uniformly affected, and deaths occurred erratically. Adaptation of the pneumonic factor to mice was accomplished from two other throat washings.

The seventeenth mouse passage was examined for its pathogenicity for puppies. Four days after intranasal injection with 0.1 ml. of a 20 per cent mouse lung suspension, 3 puppies became ill with greater severity than in natural infection. They developed a severe rhinitis, pharyngitis, and extensive cervical lymphadenopathy, and 2 died six days after inoculation. At necropsy, consolidation of one lung and a severe bronchitis and tracheitis were found. Apparently, the causal agent had increased in virulence on mouse passage. The agent was demonstrated, by mouse inoculation, in the throat washings of the sick dogs.

Filtrates were prepared from the eighteenth mouse passage of the agent by passing lung homogenates through a Selas 03 porcelain filter. Inoculation with this filtrate produced lesions in 16 of 20 mice. The filterability of the agent through other types of filters was not tested.

The age susceptibility of mice to the agent was determined (table 2). Infection has been most consistent with mice about 21 days old. Young mice appear to be refractory, but resistance disappears as they approach maturity.

Since there was a possibility that the lesions produced in the experimental mice were due to mouse pneumonitis virus (PVM), serum-neutralization tests were made by mixing the infective agent with the specific antiserum before injection. Since the disease was not prevented, it was assumed that PVM was not involved.

Other similar tests involved the use of serums from dogs recovered from tracheobronchitis and serums prepared by injecting rabbits with mouse lung suspensions containing the agent. The rabbits were given 3 to 5 injections seven days apart, and blood was collected seven days after the last injection. No neutralizing antibodies were detected,

TABLE 1—Adaptation of a Strain of the Tracheobronchitis Agent to 21-Day-Old Mice

Passage No.	Deaths	Lesions	Ratio No. infected/ No. inoculated
1	0	0	0/5
2	0	0	0/5
3	1	0	1/5
4	0	0	0/5
5	0	0	0/5
6	1	0	1/5
7	3	3	3/5
8	1	0	1/5
9	2	6	6/6
10	7	----	----
11	3	1	3/5
12	1	----	----
13	6	----	----
14	1	----	----
15	2	2	2/5
16	1	7	7/10
17	4	3	4/5
18	3	4	4/5
19	1	5	5/5

and attempts to demonstrate complement-fixing antibodies using mouse lung antigens also failed.

Infectivity titrations were made on mouse lungs by injecting tenfold dilutions into susceptible, 21-day-old mice. All $i.d._{50}$ ranged from 10^{-4} to 10^{-5} when the eighteenth mouse passage was used. Infectivity was determined by the presence of gross changes in the lungs of the mice at the time of death or when killed ten days after injection.

Chicken embryos were inoculated with mouse lung to determine whether the agent could be propagated in this medium and, if so, whether hemagglutinins would be produced. No visible changes occurred after three serial allantoic passages, and the fluids were not pathogenic for mice. No hemagglutinins were demonstrated in these fluids and no mouse lung suspensions contained

TABLE 2—The Effect of Age of Experimental Mice on Their Susceptibility to the Tracheobronchitis Agent

Age (days)	No. of mice	No. infected
2	10	0
4	12	4
6	7	6
8	7	6
21	6	6
21	5	5

hemagglutinins for chick, dog, rabbit, sheep, or human erythrocytes. Serums taken from 5 puppies during the active stages of the disease did not show cold agglutinins for human or dog erythrocytes.

DISCUSSION

The evidence to date indicates that the cause of canine tracheobronchitis is not bacterial or fungal, but that it is infectious, contagious, and of small particulate size. Presumably, the cause is a virus which dif-

fers from similar agents previously described. The virus described[2] for canine pharyngolaryngeal tracheitis was not pathogenic for mice, although it is possible that the present agent is merely a variant.

The disease is of sufficient clinical importance to warrant further investigation of its control and prevention. It is of particular significance where large numbers of animals are kept in close proximity, *i.e.*, kennels, laboratories, and animal hospitals.

Tracheobronchitis may be a problem in chronic experiments with dogs. The infection may be mild and, therefore, undetected. Nothing is known of the action of the tracheobronchitis agent on the physiology of the body systems, but the effects may be sufficient to invalidate experimental conclusions.

SUMMARY

An infective agent has been isolated from throat washings of dogs affected with tracheobronchitis. This agent, presumably a virus, is pathogenic for mice by intranasal instillation, and infected mouse lungs are infective for dogs. Certain aspects of the disease pertaining to public health and experimental animals are discussed.

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A Mammary Tumor Agent in Mice.—A ten-year study of mammary tumors in more than 4,000 female mice, at the National Cancer Institute, revealed that when young of a higher cancer strain were foster-nursed by females of a low cancer strain, the development of cancers was retarded. The reverse also was true. It is believed that cancers are incited by some factor in the milk which has many characteristics of a virus.—*Science, July 13, 1956.*

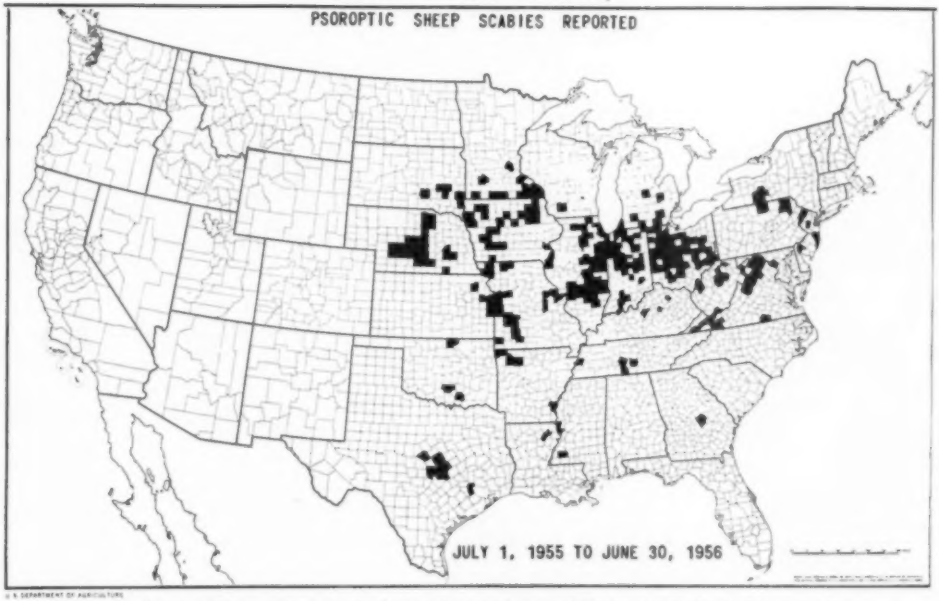
Vaccination for Fowl Typhoid.—Because of the increased prevalence of fowl typhoid, the effectiveness of several vaccines was investigated. Killed vaccines were of little or no value; those in an adjuvant of liquid paraffin and "falba" seemed to increase resistance for a few weeks. A suspension of live *Salmonella gallinarum*, attenuated by 70 subcultivations on agar at three-day intervals, induced a solid immunity in the survivors but caused the death of about one-third of the vaccinated birds.—*J. E. Wilson in Vet. Rec., Sept. 29, 1956.*

More About Dwarf Cattle.—A herd of 120 cows, all dwarfs of the six known varieties, has been assembled at the University of California. It contains Herefords, Aberdeen Angus, and Shorthorns. Less than 20 per cent of the dwarf calves live to breeding age, although delivered by cesarotomy because of the dam's small pelvis. Some can not forage and most of them are highly susceptible to bloat. Many are from 60 to 90 per cent of normal size at birth. In the short-headed Hereford type, the metacarpal bones are considerably shortened. Radiographs indicate that, in short-headed Hereford and Aberdeen Angus types, the sphenoid and occipital bones fuse within five and one-half months compared with the normal two to three years.—*Farm J., Sept., 1956.*

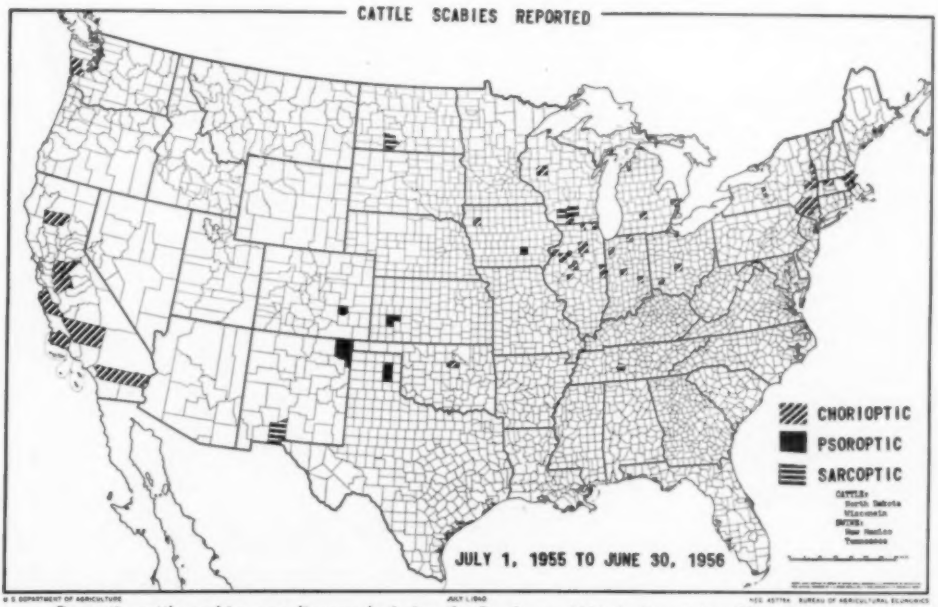
Changes in Central Nervous System of Pigs After Inoculation of Crystal Violet Vaccine.—When pigs in Germany were twice vaccinated at 14-day intervals with 5 ml. of crystal violet vaccine, then killed at varying intervals, changes in the central nervous system were observed at eight days but were greater after three to four weeks. Cells of the endothelium and adventitia of small blood vessels increased and developed into single or double layers which infiltrated the meninges of brain parenchyma. There was also focal or slightly diffuse proliferation of glia near the vessels. In pigs immunized with virulent virus and serum, the changes were similar but more pronounced, often producing encephalitis in two to three weeks. The changes following use of the crystal violet vaccine were believed due to traces of live virus in the vaccine.—*Vet. Bull., Sept., 1956.*

Scrapie Yields to No Treatment.—Six Suffolk sheep, 2½ to 3½ years old, suffering from suspected or advanced cases of scrapie, were given massive doses (1,000 mg.) of vitamin B_{12} daily for 14 days, then at increasing intervals for 56 days, without any apparent benefit. The 4 with advanced cases died during or soon after the course of treatment.—*G. N. Henderson in Vet. Rec., Sept. 8, 1956.*

Scabies in Cattle and Sheep



Psoroptic sheep scabies was reported, during the fiscal year 1956, in 607 flocks (37% more than in 1955), in 267 counties of 25 states (5 more than in 1955).—ARS, U.S.D.A., Aug. 22, 1956.



Psoroptic cattle scabies was diagnosed, during the fiscal year 1956, in five states. Chorioptic mange was diagnosed in cattle in 11 states and in cattle at four leading expositions. Sarcoptic mange was reported in cattle in two states. It was reported in swine in two states but is probably much more prevalent.—ARS, U.S.D.A., Aug. 22, 1956.

Heartworms in the Dog

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Nassau, Bahamas

Heartworm, once considered a semitropical and tropical disease, is now being found more frequently in temperate zones. Many owners returning to their northern homes after spending the winter in the South request that their dogs be blood-tested for heartworm.

Fig. 1—A dog positive for microfilaria in the blood; the only symptom exhibited was a slight eczema on both thighs.

Dogs with one or more of the following symptoms should be tested for filariasis: early fatigue, chronic cough, eczema, or anemia with heavy infestation of fleas.

One of the following quick, simple tests can be introduced as routine. Direct examination of a drop of blood obtained either from the inside of the ear flap or from a hypodermic needle inserted into a vein. The drop of blood is placed on a glass slide and examined before it dries. Positive cases will show unusually active wriggling microfilaria. At least 80 per cent of all filariasis cases can be diagnosed by this test. If the smear is negative and a diagnosis of valvular insufficiency accompanied with any of the previously mentioned symptoms seems indicated, a further blood test should be made.

Fig. 2—This dog has been affected for over four years. It shows emaciation, severe dermatitis, chronic otitis and blepharitis, cough, nephritis, dropsy, and cardiac insufficiency.

Dr. Soltys is a general practitioner in Nassau, Bahamas.

There may be a great variation in the general physical condition of dogs with heartworms. Some appear healthy with no noticeable symptoms (fig. 1), while others are anemic, coughing, itching, debilitated, dropsical, and exhausted (fig. 2). Each case must be considered according to the estimated infection and the damage that probably has occurred.

TREATMENT

In considering therapy, it must be remembered that the adult filariae have been in the blood stream for at least eight

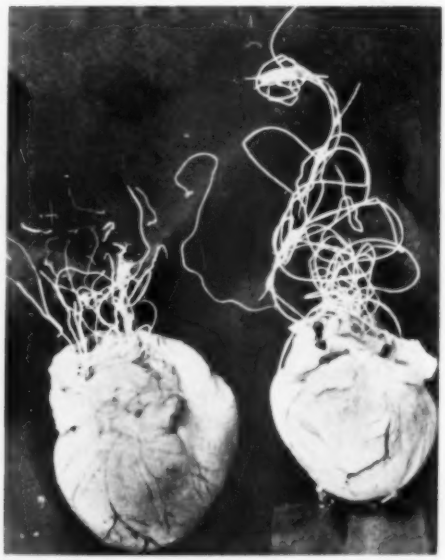

Fig. 3—The heart on the left is from a dog that has had 15 daily treatments with caparsolate sodium. Note the decomposition and breakdown of the adult parasites as compared with the healthy ones in the heart of an untreated dog on the right. The breakdown of the parasite is gradual, the threadlike decomposed end portions can be seen.

months, and the dog's heart and organs of elimination have been overworked and damaged for that minimum length of time. The first consideration is to return the patient as nearly as possible to its normal state of health. Symptomatic treatment to overcome the body's deficiency of vitamins and minerals is necessary. One satisfactory method is to feed the dog concentrated vitamins and minerals for at least two weeks prior to actual treatment. This should be continued throughout the treatment and post-treatment periods.

An attempt to hurry the treatment of filariasis to satisfy the client usually brings unfavorable results. If the filariae are killed too rapidly, materials from the dead and dying parasites (fig. 3) will result in soreness, stiffness, respiratory distress, and prostration which can be alarming. It may even be fatal. It is preferable to start the treatment with 0.5 to 1.0 cc. of caparsolate sodium* given intravenously. The patient is then returned in two days and its behavior, temperature, and heart are carefully checked for any variation. If all are normal, the dosage is stepped up to 0.1 cc. per pound of body weight, given on alternate days for 3 doses. Before each injection, the animal is checked and the client questioned as to the dog's behavior. If any signs of abnormality appear, the injections are stopped until the animal again becomes normal.

On recommencing the arsenical therapy, the dosage is given at the same level as that of the last injection. Usually a course of 15 to 18 doses of caparsolate is given. Three months after the last injection, another blood test should be made. If there is still a filarial count, a course of fuadin should be given. This usually consists of three to five injections at the rate of 1.0 cc. per 20 lb. of body weight. If the test is still positive after five injections, it may be assumed that not enough caparsolate has been given, and that live adult heartworms remain in the heart. The patient will then have to be given another course of caparsolate sodium.

Some patients can not be brought to a good enough physical condition to receive treatment even at the slow rate and, consequently, the dosage has to be reduced to as low as one half the usual rate of 0.1 cc. per pound of body weight, and the number of injections correspondingly increased.

*Produced by Abbott Laboratories, North Chicago, Ill.

If the dog, after the usual trial dose of 0.5 cc. per pound of body weight is not in a sufficiently good condition to be treated, good results can be obtained by starting the injections of caparsolate at the rate of 0.1 cc. per pound of body weight. Only three to five injections should be given. This usually results in improvement of the animal's condition probably due to the tonic effect of the arsenical. The animal should then be allowed to regain its strength and vitality for seven to 14 days or more; then six daily injections are given followed by another seven- to 14-day rest period. If no reactions occur, 2 more six-day dosages are given, with a seven-day rest period between them. Three months later, another blood test for microfilaria is made. This method of treatment is usually satisfactory.

The venom of poisonous fish is to be studied. Wounds caused by the stonefish, found in shallow waters in the Pacific and Indian oceans, produce an illness comparable to a rattlesnake bite, sometimes resulting in death; those of the sculpin, a scorpion fish, found off the coast of central and lower California, cause intense pain and swelling but no deaths have been reported from them.—*Sci. News Letter, Oct. 13, 1956.*

Mycosis Enhanced by Antibiotics.—When *Candida albicans* was injected intraperitoneally into mice, the major lesions were deep in the kidneys, indicating transmission through the blood stream. When injected intravenously, results were the same but lesions developed more rapidly. When any of the tetracyclines were given to infected mice, deaths occurred earlier and the fungus apparently penetrated the renal cortex through the peritoneum.—*Vet. Bull., Sept., 1956.*

Public Health Service Reports on Rabies.—A rabid mouse was reported (Sept. 15, 1956) to have bitten a boy on the thumb, in Kentucky. Material from the infected mouse produced rabies when injected into laboratory mice. The boy was given antirabies treatment.

Rabies in a dog only 10 weeks old was reported (Oct. 13, 1956) in Arkansas. The pup, a neighborhood pet, bit several children during one day and died the next morning. Material was not secured for laboratory confirmation.

The Use of Antihistamine (Phenergan) in Experimental Snake Venom Poisoning in Dogs

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POISONOUS snake bites occur rather frequently in domestic animals, especially in dogs. A recent questionnaire sent to all of the practicing veterinarians in Florida showed that an average of about 360 dogs are treated annually in that state for poisonous snake bites. The mortality in this group of treated dogs was 25.8 per cent.[1] Undoubtedly, many dogs die of envenomation without ever reaching the attention of the practitioner. If the incidence of poisonous snake bites in dogs in Florida is similar to that in other states (there are reasons to believe this is true, especially in the southern and southwestern states), then thousands of dogs are snake-bitten annually in the United States.

With the exception of the coral snakes, all of the poisonous snakes found in this country are pit vipers (cottonmouth or water moccasins, copperhead or highland moccasins, and approximately 26 kinds of rattlesnakes). The rattlesnakes are the most dangerous since, because of their enormous size, they produce the most venom. In general, the bites of the copperhead moccasins and the pygmy or ground rattlers are not as dangerous as those of the diamondback rattlesnakes and cottonmouth moccasins. Very few animals are bitten by coral snakes.

The purpose of this study was to determine the effectiveness of the antihistamine drug, phenergan® (promethazine hydrochloride) in treating dogs with snake venom poisoning.

The rationale for using antihistamines for poisonous snake bites is based on the observation that snake venoms cause the liberation of histamine in animal tissues.[2,3] The clinical effectiveness of antihistamine in treating snake-bitten dogs has been reported.[4] It has been demonstrated with [illegible] that phenergan antagonized the depressant action of cobra and Russell's viper venoms on blood pressure and respiration and suggested that antihistamines may be of value in the treatment of ophidiasis.[5] The inhibition of histamine activity alone by neoantergan was found to be ineffective in reducing the mortality of mice with venom poisoning.[6] Using phenergan for *Crotalus terrificus* and *Bothrops jararaca* (pit viper) poisoning in mice, it has been shown that the antihistamine drug enhanced the toxicity of the *B. jararaca* venom.[7]

The present study seemed worthwhile since antihistamines are used frequently by the veterinarians in Florida for treating snake bite in dogs, and many of these practitioners have favorable clinical impressions of the effectiveness of the drugs. Also, the reports in the literature are somewhat conflicting,[4-7] and most of the previous experimental work has been done on small animals (mice). A carefully controlled experimental evaluation in dogs should more closely approximate actual envenomation in clinical practice and help to define the efficacy of antihistamines.

Phenergan, was selected for these studies since it had been demonstrated[8] to be one of the most potent antihistamine drugs. It had also been found[9] to be more powerful and less toxic than anthisan in animals. For dogs, a dose of 25 to 50 mg., administered intramuscularly, was recommended.[10]

MATERIALS AND METHODS

Dried, crystalline, western diamondback rattlesnake *(Crotalus atrox)* venom was used in this study. It was diluted in an 0.85 per cent solution of sodium chloride so that 1 cc. of solution contained 50 mg. of venom.

The toxicity of venom samples has been found to vary[11]; hence, 3 dogs were given 2 mg. of venom per kilogram of body weight and 3 dogs were given 5 mg. of venom per kilogram of body weight to determine the amount of venom required for a lethal dose. In one experiment,[12] the minimum lethal dose (m.l.d.) for dogs had been 2 mg. of venom per kilogram of body weight,

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The authors express their gratitude to the Wyeth Laboratories, Radnor, Pa., for supplying the venom and phenergan used in this study. They also thank James G. Fish, D.V.M., Jacksonville, Fla., for the use of his animal hospital and laboratory facilities.

and in another,[13] 5 mg. per kilogram of body weight was a more effective lethal dose (l.d.) for dogs. Two of the 3 animals given 2 mg. per kilogram of body weight survived. Since the 6 dogs (control group) given 5 mg. per kilogram died, 5 mg. of our venom sample per kilogram of body weight was established as a lethal dose for dogs. All of the treated animals were given this amount of venom.

The hydrated venom was injected with a 20-gauge hypodermic needle, to a depth of ¼ inch, into the deltoid region of mongrel dogs. Three animals were given 50 mg. of phenergan intramuscularly in the opposite shoulder 30 minutes after the venom was injected, and 3 were treated in the same manner 60 minutes after the venom was injected. In addition, 3 animals were given 37 to 50 mg. of the drug intravenously 30 minutes after the venom injection, and 3 were given the same treatment 60 minutes after the venom injection. No other treatment was used and all of the experimental animals were carefully observed until death occurred.

RESULTS

Two of the 3 dogs given the smaller dose (2 mg.) of venom survived, although 1 developed an extensive slough at the injection site. All of the control animals given 5 mg. of venom per kilogram of body weight died, the survival time varying from 21 hours to 69 hours (av. 40 hr.).

The 12 animals treated with phenergan died regardless of the time treatment was instituted or the method in which the drug was administered. The survival time of the treated animals varied from 3¾ hours to 20 hours (av. 11¾ hr.). The average survival time was nine hours when phenergan was given intravenously and 13¾ hours when given intramuscularly.

The animals studied exhibited typical signs of ophidiasis. Almost immediately, the dogs experienced severe pain at the site of envenomation. Extensive edema and swelling developed in the envenomized extremity and extended to the pectoral and back regions.

Three of the animals vomited shortly after the venom was injected and 5 developed diarrhea. Four dogs exhibited excessive thirst, a sign of shock, soon after venom poisoning. In general, they preferred to lie still in their cages, and when they walked they did not bear weight on the involved foreleg because of the pain. They appeared listless, were disinterested in eating, and died before a local slough of tissue would be expected to develop.

Phenergan did not cause undue toxic effects. None of the dogs became overly excited or depressed. The only observed difference in the response of the treated versus the control animals, which could be attributed to the drug, was a transient but minimal disturbance in maintaining equilibrium. The treated animals staggered more than did the control animals when trying to walk with an injured leg.

DISCUSSION

Poisonous snake bites are dangerous in dogs and cause a high mortality; therefore, early and vigorous treatment is necessary if the animals are to be saved. In a comparative study[14] of poisonous snakes in the United States, it was determined that the poison glands of the average rattlesnake contained 220 mg. of venom; the average cottonmouth moccasin, 150 mg.; and the

TABLE I—Results of Treatment with Phenergan® in Snake Venom Poisoning in Dogs

Exper. No.	Sex	Weight in kg.	Venom (mg./kg.)	Total dose (mg.)	Treatment with phenergan	Time begun	Results
1	M	12.0	2	24.0	0*	0	Death after 57 hours.
2	M	12.7	2	25.0	0	0	Survived.
3	M	14.5	2	29.0	0	0	Survived.
4	M	13.6	5	68.0	0	0	Death after 36 hours.
5	M	8.1	5	40.5	0	0	Death after 52 hours.
6	F	10.0	5	50.0	0	0	Death after 69 hours.
7	F	13.2	5	66.0	0	0	Death after 32 hours.
8	F	11.4	5	57.0	0	0	Death after 30 hours.
9	F	8.0	5	40.0	0	0	Death after 21 hours.
10	M	10.4	5	52.0	50 mg., i. m.	30 min.	Death after 11 hours.
11	M	10.0	5	50.0	50 mg., i. m.	30 min.	Death after 20 hours.
12	M	10.0	5	50.0	50 mg., i. m.	30 min.	Death after 18 hours.
13	F	8.2	5	41.0	50 mg., i. m.	60 min.	Death after 5½ hours.
14	F	11.8	5	59.0	50 mg., i. m.	60 min.	Death after 17½ hours.
15	F	12.3	5	61.5	50 mg., i. m.	60 min.	Death after 11 hours.
16	F	9.7	5	48.5	37 mg., i. v.	30 min.	Death after 10½ hours.
17	M	11.4	5	57.0	37 mg., i. v.	30 min.	Death after 11½ hours.
18	F	9.3	5	46.5	50 mg., i. v.	30 min.	Death after 12 hours.
19	F	9.0	5	45.0	50 mg., i. v.	60 min.	Death after 5½ hours.
20	M	10.5	5	52.5	50 mg., i. v.	60 min.	Death after 3¾ hours.
21	F	10.0	5	50.0	37 mg., i. v.	60 min.	Death after 11¼ hours.

*Control animals received no drug.

average copperhead, 45 mg. If the lethal dose of rattlesnake venom for a 10-kg. dog is 5 mg. then a rattlesnake would be capable of delivering 4 to 5 lethal doses of venom. Snake bites are more serious in dogs because most dogs are bitten on the face, shoulder, or forelegs, and there is usually a time lapse of one to three hours before treatment is begun.

The crystalline rattlesnake venom used in this study was diluted so that 1 cc. of solution contained 50 mg. of venom. It takes 4 to 5 cc. of fresh, liquid snake venom to produce 1.0 Gm. of dried, crystalline venom[15]; fresh venom contains from 200 to 250 mg. per centimeter. The venom solution we used was more dilute than normal rattlesnake venom; however, this dilution seemed necessary to accurately measure the amount of venom required for a lethal dose in dogs. The venom was injected into the deltoid region as this is a frequent site of snake bites in dogs.

The inhibition of histamine activity by antihistamine drugs alone does not appear capable of reducing mortality in dogs with venom poisoning. Snake venom is a complex protein substance which has many toxic components, including proteolytic enzymes, phosphatidosis, and neurotoxins. The venom is disseminated by means of powerful spreading factors. All of the toxic effects of venom poisoning can not be attributed to histamine activity; hence, treatment with antihistamines alone would be of limited value.

All of the animals treated with phenergan from 30 to 60 minutes after envenomation died, and the treated animals died sooner than did those which remained untreated. Evidently, this antihistamine drug potentiates the toxicity of rattlesnake (*C. atrox*) venom. Animals given phenergan intravenously died sooner than did those given the drug intramuscularly. This substantiates the previous finding[7] that antihistamines enhance the toxicity of *B. jararaca* venom for mice and that a venom-intoxicated organism which just succeeds in maintaining its blood pressure in a labile equilibrium will collapse on the slightest interference, such as is provoked by the depressant action of antihistamine. In conclusion, we feel that antihistamine drugs are contraindicated in the treatment of pit viper venom poisoning in dogs.

SUMMARY AND CONCLUSION

1) Phenergan® (promethazine hydrochloride), a potent, nontoxic, antihistaminic drug, was used for treating mongrel dogs which had been poisoned by western diamondback rattlesnake (*Crotalus atrox*) venom.

2) All of the animals given a lethal dose of venom, and treated 30 to 60 minutes later with phenergan, died. The survival time of the treated animals was less than that of the untreated animals. Evidently, the drug potentiated the toxicity of the venom.

3) Antihistamine drugs are contraindicated in the treatment of snake venom poisoning in dogs.

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Case Reports of Snake Bites in Dogs

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Records of snake bites in dogs are uncommon in the Chicago area. For that reason, the following 4 cases of a condition in dogs, presumably due to snake bites, are reported.

Case 1.—On July 31, 1955, a female Boxer, 6 years old, was presented because of several small wounds on the right side of her face. She had been heard barking in the woods two hours earlier, and snake bite was suspected. Since antivenom was not available for immediate use, the wounds were lacerated in classical first-aid fashion to encourage bleeding. Calcium gluconate (20 cc.) was administered intravenously and 1 cc. of pyrahistine* was given subcutaneously.

Her pulse was 120 per minute and her temperature 101.2 F. There was extensive edema of the face and neck and pain was evident. She was given two subsequent injections of pyrahistine and, when released on the third day, most of the edema had receded and no pain was evident.

A call to the Chicago Museum of Natural History confirmed the presence, in this area, of a subspecies of rattlesnake (massasauga) whose venom is only slightly toxic, primarily containing a cytotoxin rather than a neurotoxin.

Case 2.—On June 26, 1956, at 11 p.m., the same client presented a female Boxer, 4 years old. This was 1 of 4 dogs which had "played" with a small snake approximately one hour earlier. Swelling of the lips and neck and discomfort were evident a short time afterward. There was a small, seeping wound just ventral to the right nostril, and edema of the muzzle, lips, and ventral part of the neck (resembling a dewlap) was fairly extensive. The owner also presented the dead snake, an 8-inch diamondback rattlesnake with two rattles.

Because of the previous year's experience (case 1), calcium gluconate solution was administered intravenously and no further symptoms developed.

Cases 3 and 4.—Eleven hours later, the owner brought in 2 (1 male and 1 female) more of the 4 Boxers, 1-year-old litter mates. The female had symptoms similar to, but less severe than, those described in case 2. A small (4-mm.) area of erythema, with exudate sticking to the hair and from which the hair slipped when examined, was present on the right cheek just posterior to the right commissure of the lips. That side of the face was more swollen than the other.

The male had a similar lesion on the forehead slightly to the right of the midline. The dorsum of the nose, the "stop," and eyelids were swollen, causing him to have a more worried look than usual.

Calcium gluconate (20 cc.) plus pyrahistine (1 cc.) was administered to these dogs and no further symptoms developed.

Forty-eight hours following the bite, the female (case 2) still showed slight edema of the face and neck, so pyrahistine was administered subcutaneously. The last 2 dogs (cases 3 and 4) showed little or no swelling, so all 3 were released.

The temperature and respiration of all 3 dogs remained essentially normal during hospitalization.

Shortly after the 1956 episode, the client reported killing a 2-ft. rattler, approximately 3 inches in circumference and having six rattles. Fortunately, the dogs had not tackled this one.

Dr. Cook is a general practitioner in Wilmette, Ill.

*Pyrahistine is produced by Pitman-Moore Co., Indianapolis, Ind.

A Man Not Immunized by Snake Bite

A professional herpetologist, who milks poisonous snakes for their venom, was bitten ten times in 12 years. Each time he identified the snake and recorded his subjective reactions. The two most serious reactions were to the bite of the eastern diamondback rattlesnake, the most deadly snake in this country. The last of these bites was in the left thumb. He applied a tourniquet to the wrist, cut his hand open in three places, then was driven 27 miles to a hospital. In 15 minutes, he could feel the effects of the venom; in 30 minutes, he was unable to move hands or legs; and in 45 minutes, he was totally helpless.

In spite of heroic treatment, in five hours his arm was twice its normal size and in 24

hours, the swelling had spread to the scapular, pectoral, and cervical regions. In five days, his condition began to improve but he was unable to resume activities for six weeks. The thumb gradually healed but is permanently atrophied.

Of the 12 bites, three were severely toxic, two were moderately toxic with extensive local effects, and five had only minimal local effects. This patient gives no evidence of either immunity or sensitivity to snake venom as the result of previous snake bites. —*J.Am.M.A., July 7, 1956.*

The Use of Antivenin for Rattlesnake Bite in a Cat

HARRY M. KROLL, D.V.M.

Dunmore, Pennsylvania

A 3-year-old cat was struck in the carpometacarpal region by a rattlesnake. The owner witnessed the incident. There was immediate swelling in the area with a slight trickle of blood from the wounds. Within a half hour, emergency measures were directed by telephone. A tight tourniquet was properly applied, but the owner did not incise the wounds.

A search for antivenin was successful and it was given exactly two hours after the cat was bitten. The product* was a hyperimmune polyvalent North American anti-snake bite equine serum containing antibodies against venom of the rattlesnake, copperhead, and moccasin. The dose used was one package of 15 cc. of reconstituted dried serum.

When the cat was admitted to the hospital, the tourniquet was left in place, deep cruciate incisions were made to allow the escape of blood and venomous deposit, and a few cubic centimeters of antivenin serum were injected around the bite wounds. The remainder was given subcutaneously. The cat showed no signs of discomfort or malaise. Recovery took place promptly.

This is the author's first experience with snake bite. It is reported since others might wish to familiarize themselves with the problem.

[The following information was secured from a veterinarian who has treated a number of cases of snake bite in dogs and who has produced snake bite toxicity by injecting the venom into well dogs. He reports that one drop of fresh venom contains 60 mg. of toxin when in the crystalline form, and that 1 mg. per pound of body weight constitutes a lethal dose for a dog. One vial (15 cc. when reconstituted) of antivenin is said to neutralize 10 mg. of venom, which means that six vials would be required to neutralize one drop of venom.

The severity of the bite depends on the amount of venom injected. A 5-ft. rattlesnake can be milked of 3 cc. of venom (2,700 mg. of crystalline toxin) but the amount usually deposited per strike is not known. The bite of a snake 2½ ft. long is said to seldom cause death in a dog, but the dog will be critically ill for 12 to 18 hours. Venom is absorbed slowly, therefore, if a tourniquet is applied promptly, the wound widely incised, and suction applied for several hours, the animal can usually be saved. The use of cortisone* and blood transfusions will usually prolong the life of an animal until the venom can be neutralized by the body defenses. Many experienced practitioners seldom use antivenin because the amount required for effect may cause a serious serum reaction.

The JOURNAL will welcome further information on the snake bite problem.—W.A.A.]

Dr. Kroll is, at present, engaged in small animal practice in the Scranton, Pa., region.

*The antivenin was a product of Wyeth and Co., Philadelphia, Pa.

*Oral doses of 25 mg. of cortisone every six hours is recommended for snake bite in children (J.Am.M.A., May 16, 1953).

Leptospira canicola infection was reported in six dog owners in Berlin. Three of the dogs had titers up to 1:6,400 against *L. canicola*. The other 3 had been killed without test.—*Vet. Bull., Sept., 1956.*

Sublingual Petechiae in Equine Infectious Anemia.—An examination of 306 horses, 48 of which had infectious anemia, revealed typical sublingual blood spots in most of them. However, only 5 per cent of the anemia-free animals had 50 or more petechiae, whereas these were present in 67 per cent of the horses with anemia. These lesions are of value in clinical diagnosis, but they are not pathognomonic. When they occur, a liver biopsy or a transmission test, or both, should be performed. —*German Sci. Bull No. 1, July, 1956.*

What Is Your Diagnosis?

Because of the interest in veterinary radiology, a case history and accompanying radiographs depicting a diagnostic problem are usually published in each issue of the JOURNAL.

Make your diagnosis from the picture below—then turn the page ▸

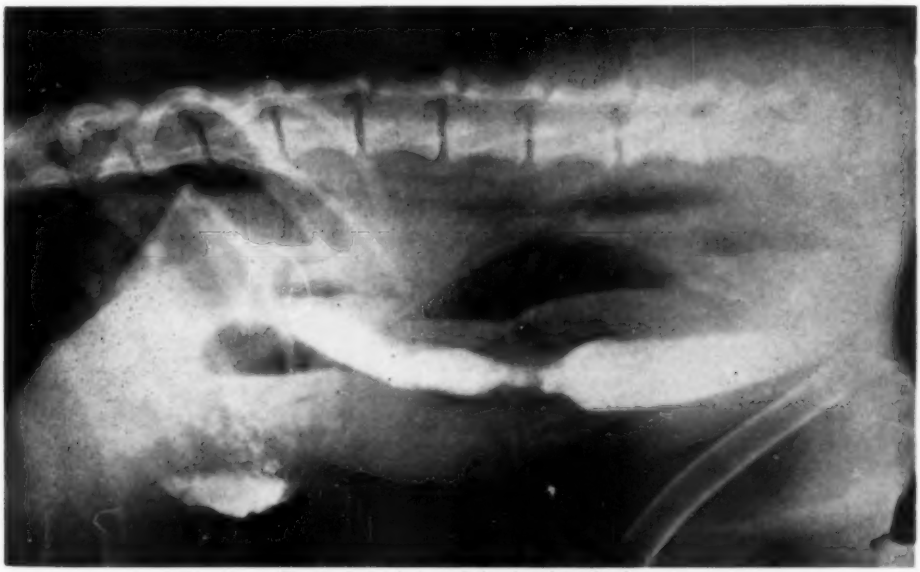

Fig. 1—Lateral view of the dog (standing).

History.—A male Boxer, 4 years old, had been losing weight and vomiting periodically for three weeks. Three ounces of barium solution was given by mouth and a radiograph was taken two hours later.

(Diagnosis and findings are reported on the next page)

Here Is the Diagnosis

(*Continued from preceding page*)

Diagnosis.—Obstruction of the small intestine.

Editor's Comment.—Several diagnostic signs pointed out by Carlson (JOURNAL, March 1, 1956, p. 246, fig. 5) appear in this radiograph. In intestinal obstruction, gas becomes trapped over the solid contents of the intestine and shows characteristically in radiographs taken with the dog in a lateral standing position.

In intestinal foreign body obstruction, the portion of the bowel through which the foreign body has passed may be greatly damaged. The fatigued bowel may be atonic and dilated.

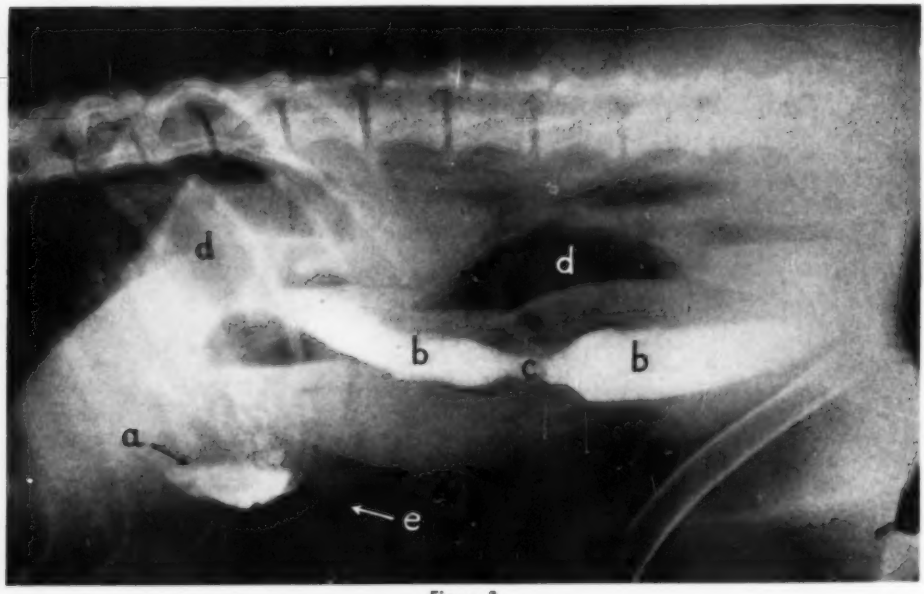

Figure 2

Notice in figure 2 that the stomach (a) is atonic (barium in the organ); also that the dilated small intestine (b) is contracted at one point (c), with gas pockets (d) in some loops. The circular object (e) at the posterior rib margin proved to be a walnut, the cause of the obstruction.

This case report was submitted by Dr. Julius J. Fishler, Elkhart, Ind.

Our readers are invited to submit case histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

Use of Furazolidone in a Field Outbreak of Salmonellosis in Mallard Ducks

F. R. LUCAS, V.M.D.

Centreville, Maryland

"KEEL" DISEASE, or salmonellosis, in ducks was first described in 1918 by Manninger, who isolated a type-B paratyphoid organism from diseased birds. Later investigations revealed that several species of Salmonella might be involved, young birds being especially susceptible. Mortality among infected flocks may be high and has been known to reach almost 100 per cent. The main symptoms are drowsiness, anorexia, intense thirst, occasional diarrhea, and the characteristic stagger, stretch, and "keel-over" just before death, whence came the name "keel" for the disease. Biester and Schwarte[1] state that "little is known regarding the occurrence of paratyphoid infection in wild ducks."

This study is presented for its two interesting aspects which are salmonellosis in a group of mallard ducks and the results of treatment with furazolidone.

Furazolidone* is a synthetic nitrofuran compound, endowed with potent antimicrobial action.[2] Although the drug became generally available only a few years ago, there are a number of published studies about it in basic and clinical fields of veterinary medicine. Gram-negative bacteria are generally very susceptible to furazolidone[2] and all 58 tested strains of ten species of Salmonella were found uniformly sensitive *in vitro*.[3] In salmonelloses of poultry, the encouraging results reported in experimental studies with chickens[4] and turkeys[5] have been confirmed in field studies of naturally occurring infection in these birds[6,7]; however, the use of furazolidone in either wild or domestic ducks has not, to the author's knowledge, been previously reported.

The field study reported here was conducted by the author on a 3,000-acre farm, situated on the eastern shore of Maryland, devoted wholly to a breeding and conservation program for mallard ducks and geese. Numerous ponds covering large areas served to attract "visitors" among the wild geese and ducks, as well as to provide watering space for these birds. The ration provided for the birds consisted of a nationally advertised commercial turkey starter. Housing was ideal and adequate. Each house had a concrete floor, in one part of which was a pond, 2 ft. deep, 3 ft. wide, and 30 ft. long; the water was changed daily. Brooding was accomplished by ample infrared lamps. After the ducklings were 6 weeks old, they were transferred to the outdoor ponds.

Hatching was done in two modern Petersine incubators with forced-draft ventilation and a capacity of 7,000 eggs. The eggs were collected daily, during the breeding season, from nail keg nests set on posts located around the perimeter of a shallow outdoor breeding pond. They were held in storage until a sufficient quantity (4,000 to 5,000) were accumulated for incubation. Hatching normally required 28 days, and hatchability was estimated at 55 to 60 per cent. Six hatches were completed during the 1955 season. No effort to fumigate the incubator was made during this first season's attempt at artificial hatching of mallard ducks. The first four hatches were apparently healthy with no appreciable loss; however, at the time the first specimens were submitted, losses ranged from 40 to 60 per day. Of 16,000 ducklings hatched during the 1955 season, approximately 2,000 birds (12%) were lost.

CLINICAL MANIFESTATIONS AND COURSE OF THE DISEASE

On June 9, 2 ducklings, 1 week old, and on June 21, 4 ducklings, 6 weeks old, were presented to the laboratory for diagnosis. Symptoms were similar to those previously described by Rettger and Scoville:[8]

The affected individuals appeared weak and sluggish and remained close to the heating pipes. They were not easily aroused and did not go in

From the Live Stock Sanitary Service Laboratory, Maryland State Board of Agriculture, Centreville.

*Furazolidone is the generic name of the drug N-(5-nitro-2-furfurylidene)-3-amino-2-oxazolidone.

Acknowledgement is made to the following individuals for assistance: Dr. P. R. Edwards, Enteric Disease Laboratory, Chamblee, Ga., for typing of cultures; Eaton Laboratories, Norwich, N.Y., for supplying furoxone; Dr. J. O. Alberts, University of Illinois, Urbana, for testing serum for viral hepatitis and Newcastle disease; and Dr. P. A. Hansen, livestock laboratory, College Park, Md., for preparation of specific antigens used in test.

search of food as did the others. They ate at times, however, and their crops were distended. One of the marked symptoms was intense thirst. After drinking, some of the ducklings drew themselves to full height, staggered a few seconds, keeled over and after one or two gasps died — hence the name "keel" for the disease. Examination of the dead ducklings revealed no pathological lesions except, perhaps, general paleness of the tissues and low body weight.

The disease apparently was spread by the last two hatches to other groups of ducks. This spread was attributed to inability to control migration of birds from one area to another and comingling of birds of all age groups.

The period of time covered in this report included June to October, with frequent visits personally made to assist in controlling the loss of ducks as well as spread of the infection.

LABORATORY TESTS

Sections of approximately 1 Gm. of heart, liver, ovaries, spleen, and lungs, from ducks obtained either on personal visits or brought to the laboratory, were inoculated into tetrathionate broth and incubated for 24 hours. Plates were prepared from broth cultures and typical colonies were selected and inoculated on T.S.I. agar slants for 24 hours. A loopful of this material was then inoculated into urea broth and sugars for fermentation studies. A diagnosis of paratyphoid was made,[9,10] since the cultures were urease negative and formed acid and gas in dextrose, maltose, and xylose, and showed motility in SIM mediums with H_2S present. Serotypings of cultures obtained from bacteriological studies were identified by the U. S. Public Health Service as *Salmonella typhimurium* and *Salmonella anatum*.

TREATMENT

In view of the paratyphoid infection found to be present, treatment with 1.5 lb. of furazolidone premix per ton of ration (0.00825% furazolidone) was advised in a preliminary effort to ascertain dosage level. After four days' use of furazolidone at this level, losses were reduced from 40 to 60 ducklings per day to 15 to 20 per day. At this time, furoxone concentrate veterinary®† and a level of 60 oz. of concentrate per ton (0.011% furazolidone) was fed.

This higher concentration cut the losses to 1 to 4 birds a day. During one of our inspection trips, it was decided to gather all visibly sick birds (100) into a hospital pen and treat each with furoxone tablets (100 mg.) *per os*. These birds were about 4 to 6 weeks old and weighed 2 lb. or less. On the first day of treatment no adverse effects were noticed; however, after treatment the following day, the caretaker observed that, within 15 to 30 minutes, the birds would suddenly jump up, run blindly into fences or other objects, whirl around in circles, fall down and, after lying motionless for a time, would eventually arise and act normal. It was, therefore, recommended that the dose be reduced to 50 mg. every other day for a total of four doses. About 60 of these birds died within several days after isolation. Ten days later, all but 4 had died, probably due to dehydration and starvation in conjunction with severe debilitation from the effects of the disease.

To determine the effectiveness of the drug, a control attempt was made by withholding medicated feed after 14 days of treatment with 0.011 per cent furazolidone in mash. A group of several thousand birds, all on one pond, was placed on regular unmedicated mash. Within two days, losses had climbed from 2 to 3 per day to 20 to 30 per day. In view of the increased losses, furazolidone was then continuously fed to all younger birds (16,000) for a six-week period.

On August 29, blood samples were taken from 480 breeders. A tube-agglutination test for salmonellosis resulted in 12 positive reactions (2.5%) using regular pullorum antigen,[11] and in 16 (3.3%) using Wright's polyvalent pullorum antigen. Bacteriological examinations of three reactors were positive for *S. anatum*. Pooled samples of serum were negative for Newcastle disease and viral hepatitis.

On October 10, a tube-agglutination test, using specific antigens against *S. typhimurium* and *S. anatum*, was made on 100 breeders. These tests with *S. anatum* (flagellar) antigen gave 14 positive reactions, while the *S. anatum* (somatic) gave 13 positives. The *S. typhimurium* (flagellar) gave five positive reactions, with no reactors to *S. typhimurium* (somatic).

DISCUSSION OF FINDINGS

As a result of these tests and bacteriological findings, it can be surmised that the infection could have occurred as the result

†Trademark of, and supplied by, Eaton Laboratories, Norwich, N.Y.

of infected eggs from carrier birds being hatched and further contaminating other eggs present in the incubator.[12,13] This agrees with the findings, in 1936,[14] that the disease takes a chronic course in the adult duck with signs of oophoritis (orchitis in the male). Infected eggs are laid, producing diseased ducklings which generally soon die from an acute septicemia; survivors are carriers which produce infected eggs. During its formation, the egg is infected from the diseased ovary or oviduct, but it can also be externally infected by the feces or by contact with an infected nest or incubator. Hen eggs may also be of importance on infected farms, as was observed[14] in an outbreak of *Salmonella enteritidis* among chicks. The elimination of infection from an infected farm necessitates strict hygienic measures and the isolation of suspected and diseased birds, when detected by the rapid serum-agglutination test once a year. A mild positive reaction should always be taken as a definite indication of infection.

Concerning the transmission of this disease, infection from the egg-laying organs is more important than fecal contamination in the cloaca, which emphasizes the urgent need for a method of systematic examination of blood and feces from duck farms to cope with the disease.[15]

In addition to specific treatment as indicated, fumigation of the incubators during hatching is highly recommended, in conjunction with bleeding and testing of breeders, to eliminate possible carriers.

SUMMARY

A severe outbreak of "keel" disease on a farm artificially incubating mallard duck eggs caused the loss of about 2,000 mallard ducklings, 1 day to 4 weeks old, out of a total of 16,000 hatched during the 1955 season, before the disease could be brought under control. *Salmonella anatum* and *Salmonella typhimurium* were isolated from diseased birds, and blood-agglutination tests on about 600 breeders were positive in up to 14 per cent of the birds.

Treatment with furazolidone at 0.00825 per cent in the feed reduced losses from about 50 to 18 per day; an increase in dosage to 0.011 per cent effectively controlled the disease. Individual treatment of 2-lb. ducks, 4 to 6 weeks old, with 100 mg. of furazolidone daily in tablet form, produced toxic signs, but 50 mg. daily was well tolerated. The diagnosis, mode of transmission, and treatment are discussed.

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Trypsin for Trauma or Inflammation.—When the enzyme, trypsin, is given parenterally to patients with trauma or inflammation, marked reduction in the edema, blood stasis and pain usually follows. Trypsin tablets (5 mg.), to be slowly absorbed in the mouth, have recently been proved effective in man when given every four hours.—*Antibiot. Med. and Clin. Therap., Sept., 1956.*

Feed Type and Rumen Development

When 46 newborn calves were fed various rations and slaughtered at different ages up to 16 weeks (at Cornell University), the four compartments of the stomach were found to develop uniformly on an all-milk ration. However, when grain or roughage or both were fed, the nonglandular compartments developed more rapidly than the abomasum. The abomasum development, in general, was in proportion to the body weight.

While the rumen had no prominent papillae at birth, some were present at 4 weeks in milk-fed calves but their height (2 mm.) increased little in 16 weeks. (Milk usually goes directly to the abomasum.) However, papillary growth was noticeable at 4 weeks and marked at 7 weeks (1 cm.) when either grain or hay were fed. Grain contains much less food fiber (7%) than does roughage, indicating that papillary development may depend more on the chemical than the physical qualities of the food. One calf, fed no dry material except 1.5 lb. of nylon bristles when 10 days old, had no papillary growth when the bristles had been in the rumen 70 days. Since nutrients are absorbed in the rumen and reticulum and since papillae increase the absorptive surface, their development probably indicates a response to the production of soluble products by the microbiological activity. —*J. Agric. and Food Chem., Sept., 1956.*

Effect of Cobalt on Rumen Digestion.—The *in vitro* cellulose digestion by rumen microorganisms was slightly depressed by the addition of 12 p.p.m. of cobalt, strongly depressed by 100 p.p.m. Addition of vitamin B_{12} had no appreciable effect. *In vitro* digestion of cellulose was also depressed by inoculum taken from a steer fed high levels of cobalt. This depression was not as great when urea had also been fed.—*J. Anim. Sci., Aug., 1956.*

Copper Deficiency and Enzymes

In copper-deficient animals, there is an impairment in the absorption of iron, in mobilization of tissue iron, and in utilization of parenterally administered iron. However, some signs of copper deficiency are not related to iron metabolism; copper-deficient pigs develop a condition resembling ricketts with atrophy of leg muscles, and copper-deficient sheep show deterioration of the fleece and neurological disturbances in the offspring.

In a copper-deficiency experiment with rats, growth was reduced after three weeks, anemia developed in ten weeks, and deaths occurred after 14 weeks. Of the 14 enzymes studied, only cytochrome oxidase showed a marked decline in quantity; the other iron-containing enzymes were not depressed. The exact mechanism of the action of copper in the synthesis of the iron-porphyrin ring remains unknown.—*Nutr. Rev., Sept., 1956.*

Acute Ergot Poisoning in Steers

In late summer, 1954, in Natal, South Africa, 23 steers, 2 years old, were placed in a pasture which had been rested for several months. The grasses had ripened and one of them was heavily infested with ergot (*Claviceps purpurea*). In 24 hours, 1 steer was lying apart, had difficulty in rising, and slight muscular incoordination. At 72 hours, most of the steers were lying separately, appeared nervous, rose with difficulty, had marked muscular incoordination (1 fell after rising), and showed signs of aggressiveness toward each other. They were hypersensitive, their ears and skin twitched, their eyes were staring and anxious, and they showed marked ataxia. They seemed hungry but had difficulty in maintaining their balance when the head was lowered.

When moved to an ergot-free pasture, the steers showed improvement in a few hours. In 48 hours, all had recovered except 2 that were badly affected. They lost some condition and their coats became harsh during the two weeks it took them to recover completely.

This pasture had been continuously grazed during the spring and early summer by pregnant cows and heifers without incident.—*J. South Afric. Vet. Med. A., June, 1956.*

Antibiotic Concentration in Eggs.—Laying hens, 1 year old, were fed aureomycin® at levels of 50 to 2,000 Gm./ton of feed. The antibiotic was not detected in the eggs of hens fed less than 200 Gm./ton, but at the 2,000 Gm./ton level they contained 0.141 μg./Gm. of egg. The eggs laid one week after the antibiotic was removed from the feed were negative.—*Poult. Sci., July, 1956.*

A Re-evaluation of Postmortem Examinations

In medicine, in recent decades, there seems to have been a greater shift in the evaluation of postmortem examinations than has occurred, we hope, in veterinary medicine. An editorial writer in the *Journal of the American Medical Association* (March 31, 1956) recalls the generation when medicine was dominated by the school of pathologists; when young physicians flocked to Europe where an autopsy was required on every patient that died in a hospital. A comparison is made with medical education today, when an autopsy is often considered a burden.

In the former era, the autopsy room was said to be the center of research, and the clinical conferences conducted there were considered "the last word in undergraduate teaching." The strength of the system was its insistence on demonstrable fact which helped to sweep away "a host of medical vagaries." It also aided in eliminating the trust in illogical remedies, but it admittedly produced few positive advances in therapeutic measures. If evidence of disease was not found, the condition was "likely to be dismissed as imaginary." During that era, interest in preventive medicine and experimentation was at low ebb.

The present era is said to present a distinct contrast, characterized by reliance on experimentation, by interest in the functional aspects of disease, and by advances in therapeutics. Autopsies are still considered important in teaching and in determining the end results of surgery, but for little else. To revive interest, it is suggested that the system be simplified so stacks of autopsy records will not accumulate, to wait in vain for analysis; also, that the records and microscopic sections be restricted to items of immediate value to the clinicians involved, and that autopsies be performed, "as in the old days," by the physicians interested in the case.

According to the editorial, certain materials from the cadaver often are of more value for experimentation than similar materials from experimental animals. Experiments with autopsy material, such as surgical repair of abnormal heart valves or testing the tensile strength of an aneurysm, are suggested.

If the autopsy is being neglected, has the pendulum swung too far? Veterinary medicine has profited tremendously from following the lead of medicine but to follow in a de-emphasis of the necropsy would be tragic. However, new developments should be watched and useful features adopted.

Necropsies in Veterinary Medicine

The necropsy is essential in diagnosing diseases of the herd or flock.

Furthermore, in any branch of medicine, nothing is as important as a correct diagnosis, and nothing sharpens the ability to make a correct one as much as regular follow-ups on treated cases, alive or dead. Also, nothing builds a client's faith more than telling him what to expect if and when the patient dies, and then being able to demonstrate those changes upon necropsy.

Actually, veterinary medicine, as compared with medicine, has never taken full advantage of the necropsy. Too often, pathology has been considered merely as a basic subject, such as anatomy.

The clinical picture must be correlated to the necropsy lesion—*i.e.*, a shadow on a radiograph with a lesion in the lung, or signs of arthritis with lesions in the joint. For example, when a pathologist was alerted to the existence of a certain transmissible lesion, he found that the clinicians in his area considered them too unimportant to submit for identification. At another clinic the significance of several less common lesions was not appreciated until an inquisitive pathologist initiated thorough studies.

These instances indicate the value of complete, systematic necropsies and, also, the importance of teamwork between clinicians and laboratory diagnosticians.

Vibriosis Not Spread by Ram

Venereal diseases are of great significance to breeders of animals. That *Vibrio fetus* infection could be transmitted by the breeding bull, or his semen, has long been recognized but that the ram played a similar role seemed doubtful. An article titled "Experimental Vibriosis in Sheep" (p. 511) confirms previous evidence that this is not a venereal disease in sheep. This species peculiarity somewhat parallels the phenomenon in brucellosis, which is rarely a venereal disease in cattle but frequently may be in swine.

Current Literature

ABSTRACTS

Encapsulation of Heartworm in Mosquitoes

Data are presented on the encapsulation (chitinization) of the dog heartworm, *Dirofilaria immitis*, in *Aëdes aegypti*. Encapsulation occurred mainly on microfilariae and rarely on "sausage stage" larvae. Both the intensity of encapsulated parasites and the number of mosquitoes with encapsulated forms increased with time spent by the parasites in the mosquito. Similarly, the percentage of completely encapsulated filaria larvae increased as their stay within the mosquito lengthened.

The data suggest that, in an unfavorable mosquito host, encapsulation of microfilaria-like larval parasites was favored in direct relation to the length of time spent in the host.—[*Leo Kartman: Notes on the Encapsulation of Dirofilaria Immitis in the Mosquito Aëdes Aegypti. Am. J. Vet. Res., 17, (Oct., 1956): 810.*]

Spinal Immobilization Methods in the Dog

Methods of spinal immobilization by means of bone grafts were studied in 14 dogs. The Albee (wedge or inclusion) graft on the spinous processes apparently gave the best immobilization and fusion of the vertebrae involved. It was further determined that some additional support to the back itself is desirable for best results.

Other means of spinal immobilization were investigated in 8 dogs in which simulated fractures had been produced. The results showed that plates and bolts applied to the spinous processes were excellent, except in dogs that were young or so large that stresses applied to the plate might break the bone process. Three clinical cases of spinal fracture corrected by the above method are reported.—[*B. F. Hoerlein: Spinal Fusion and Vertebral Immobilization Methods in the Dog. Am. J. Vet. Res., 17, (Oct., 1956): 695-709.*]

Effect of Octin on Gastroenteritis in Baby Pigs

Observations were made of the effect of octin (isometheptene, methyliso-octenylamine HCl) on the course of transmissible gastroenteritis (TGE) in 16 pigs, 4 to 6 days old. Octin added to the milk at feeding time at the rate of 1/25 gr. and 1/12 gr. had no significant effect on the course of the disease under the conditions of these experiments.

Octin was administered intramuscularly to TGE-infected pigs, approximately 4 weeks old. The administration of octin two hours after feeding had no effect upon the retention of sodium, potassium, and nitrogen. The administration of octin to TGE-infected pigs at feeding time produced vomiting and decreased their retention of sodium, potassium, and nitrogen. Octin does not appear to have therapeutic value in the treatment of pigs infected with TGE.—[*Elwood F. Reber and Paul D. Beamer: The Effect of Octin on Baby Pigs Infected with Transmissible Gastroenteritis Virus. Am J. Vet. Res., 17, (Oct., 1956): 643-645.*]

Tumors in Cattle, Sheep, and Swine

A two-year survey of tumors occurring in cattle, sheep, and swine, submitted for slaughter at federally inspected abattoirs in Denver, was conducted and 1,002 tumors (908 bovine, 66 ovine, and 28 porcine) were collected. Of the 908 bovine tumors, 722 were squamous cell neoplasms of the eye and were found in 532 cattle during the first year of the survey. The collection of eye tumors was limited to those found in selected abattoirs during one year; their frequent occurrence made them the most important neoplasm encountered in cattle. A diagnosis of squamous cell carcinoma was made in 471 of the 722 eye tumors. All other tumors in the three species detected by inspectors were collected for the entire two-year period.

Lymphosarcomas and adenocarcinomas of the uterus in cattle were economically important because of their comparatively frequent occurrence and predictable early fatal termination. Schwann's cell and adrenocortical tumors were common bovine tumors with a relatively benign behavior.

Neoplastic diseases in slaughtered sheep and swine are much less important than in cattle. Epithelial tumors were approximately equal in occurrence to the nonepithelial tumors in sheep but were not found in swine in the survey. Lymphosarcomas would appear to be the tumor of the greatest economic importance in both sheep and swine. Embryonal nephromas were the most common tumor diagnosed in swine.—[*A. W. Monlux, W. A. Anderson, and C. L. Davis: A Survey of Tumors Occurring in Cattle, Sheep, and Swine. Am. J. Vet. Res., 17, (Oct., 1956): 646-677.*]

Mouse Pox in the United States

Mouse pox is one of the most important diseases threatening mouse colonies in this country today. The first sign of the disease is the high mortality without apparent cause or any external lesions. Less acute forms are characterized by necrosis of the extremities. Pocklike lesions are not seen unless the animal survives until the ninth or tenth day after infection. The disease may be controlled by vaccination.—[*B. A. Briody: Mouse Pox (Ectromelia) in the United States. Proc. Animal Care Panel (1955): 1-8.*]—N. R. BREWER.

Immunity from Newcastle Disease Virus Vaccine

Measurement of immunity in chickens vaccinated twice with inactivated Newcastle disease virus (NDV) vaccine was attempted by employing a potency test in which the challenge virus is titrated in both the vaccinated and the control chickens. The difference in titers between the vaccinated and the control chickens represents an index of the degree of immunity. The intranasal, intramuscular, and aerosol methods of challenge

were employed. Infection in the titrations was based on virus recovery from the blood at 96 hours following challenge. After this, the controls were destroyed and the vaccinated birds observed for an additional ten days.

Following intranasal challenge, the chicken-infective titer of the virus in the vaccinated birds was approximately 10^3, while in the controls it was 10^6. Thus, the vaccinated birds were able to withstand 10^5 chicken-infective doses by the intranasal route. Following intramuscular challenge, the virus-infective titer in the controls was $10^{5.5}$; however, in the vaccinated birds, the titer could not be determined. The aerosol exposure resulted in a titer of $10^{5.4}$ in the controls, while in the vaccinated chickens the titer was estimated to be $<10^1$.

None of the vaccinated chickens in the three groups became clinically ill or died in the ten-day observation period after challenge. Serum antibody testing revealed a substantial antibody titer by both the serum-neutralization and the hemagglutination-inhibition tests.—[*M. S. Hofstad: Further Studies on the Evaluation of Immunity in Chickens Vaccinated with Formalin-Inactivated Newcastle Disease Virus Vaccine. Am. J. Vet. Res., 17, (1956): 738-741.*]

Aplastic Anemia in Chickens Fed TCESOM

Young chickens fed semisynthetic rations containing 72.9 per cent trichloroethylene-extracted soybean oil meal (TCESOM), 72.9 per cent hexane-extracted soybean oil meal (HEXSOM), or a natural corn-soybean ration were inoculated with suspensions of avian lymphomatosis tumors. There was no difference in the development of tumors in the chickens on the different rations.

The chickens fed TCESOM developed aplastic anemia with clinical signs and hematological and pathological changes indistinguishable from those in chickens with field cases of aplastic anemia or so-called hemorrhagic syndrome. No blood changes occurred in those fed HEXSOM or the natural rations.—[*W. R. Pritchard, O. S. Davis, D. B. Taylor, and L. P. Doyle: Aplastic Anemia in Chickens Fed Trichloroethylene-Extracted Soybean Oil Meal (TCESOM) and Failure of this Dietary Meal to Suppress the Development of Experimental Lymphomatosis. Am. J. Vet. Res., 17, (Oct., 1956): 771-777.*]

Palatability Tests for Cats

Tests for the screening of palatability of ration-fed cats have been described. In most instances, a test utilizing 15 adult cats over a two-day period was sufficient to differentiate between two rations. Rank analysis and *chi* square analysis were found to be more satisfactory than standard statistical procedures in analyzing this type of data.

Since some animals had definite food preferences that were different from the majority, the selection of the animals used may be crucial. In general, there was a tendency for the cats to choose a new ration rather than the one which they were usually fed. Also, when the animals were offered two foods over a period of time, their preference sometimes changed significantly. Certain foods of high acceptability were nutritionally inadequate, while some foods that appeared nutritionally adequate had a low acceptability when offered in the presence of other rations.—[*Mark D. Hegsted, Stanley N. Gershoff, and Eugene Lentini: The Development of Palatability Tests for Cats. Am. J. Vet. Res., 17, (Oct., 1956): 733-737.*]

BOOKS AND REPORTS

Veterinary Ophthalmology

There has been an urgent need for a text on veterinary ophthalmology for a generation past. For this reason, the book is certainly welcomed. It is evident, however, that the author has written a treatise to appeal to zoologists, physiologists, anatomists, teachers, research workers, and clinicians. With this information, gleaned and composited from many sources, the book fulfills a need for many in the allied fields, and yet, therein lies its weakness.

The clinical material occupies but one half of the contents and considers both large and small animals, thus further diluting the discussion. The description of diseases, treatment, and surgical procedures are not afforded adequate space. The chapter on eyelids and lid surgery is especially well done and with good illustrations, but little to no attention is given glaucoma and cataract procedures. Treatments differ somewhat from those in common use in this country, but in this respect much may be learned.

One chapter adequately discusses conditions which affect the retina and optic nerve; however, a few illustrations would have enhanced the discussion. The dearth of information on this subject in the American literature makes this the most valuable chapter in the book.

The author is to be commended for helping to overcome a deficiency in our knowledge of ophthalmology. He admits that the book is but a beginning and lacking in many respects, but hopes that fresh interest will be aroused in an important but hitherto neglected subject.—[*Veterinary Ophthalmology. By R. H. Smythe. Williams and Wilkins Co., Baltimore 2, Md. 1956. Price $7.50.*]—W. G. MAGRANE.

Fertilization

The subject of this little book is "the life of the egg from the attachment of the fertilizing spermatozoon to the fusion or apposition of the male and female pronuclei."

The text is technical and has little practical value for veterinarians.—[*Fertilization. By Lord Rothschild. 170 pages, Illustrated. Methuen and Co. Ltd., London, and John Wiley and Sons, Inc., New York, N. Y. 1956. Price $3.50.*]—H. E. KINGMAN, SR.

THE NEWS

Enrollment in Veterinary Schools, 1956-1957

Data on the students enrolled in the 17 Schools of Veterinary Medicine in the United States and the 2 in Canada for the present year have been compiled and the totals are compared with those of last year (table 2).

Also shown (table 1) is the relative amount of preprofessional training of the 942 first-year students (freshmen) at the 17 schools in the United States. This new class contains 207 students who already have bachelor degrees and 11 with master degrees. Those enrolled were selected from approximately 2,300 qualified applicants, a ratio of 1:2.5, the same as in recent years.

TABLE 1—Preprofessional Training, U. S. Schools

Freshmen	Years of preveterinary training: 2 yr. (%)	3 yr. (%)	4 yr. or more (%)	Degrees (%)
1952-1953	42	25	33	25.0
1953-1954	41	26	33	25.8
1954-1955	47	23	30	25.8
1955-1956	45	24	31	26.6
1956-1957	45	28	27	21.2

A comparison with 1955 shows that the 1,023 first-year students in the 19 schools is an increase of 11, the total enrollment of 3,958 is an increase of 18, and the 181 veterinarians taking graduate work is an increase of 2.

Of the 3,754 undergraduate students, 84 (2.24%) are women. The class distribution of the 84 is freshmen, 23; sophomore, 16; junior, 29; and senior, 16.

Veterinary Faculty Changes

The following changes in veterinary personnel in the faculties at the Schools of Veterinary Medicine were reported at the beginning of the 1956-1957 school term:

Alabama.—The following veterinarians have been appointed to the staff of the School of Veterinary Medicine, Alabama Polytechnic Institute: D. F. Johnson, Jr. (TEX '44), professor of pathology; William E. Ribelin (ISC '49), professor of pathology; R. P. Shields (API '56), instructor, clinical pathology; Lawrence E. Evans (KSC '51), professor, anatomy and histology; T. W. Weatherford (API '54), instructor, anatomy and histology; Howard P. Hobson (ILL '56), instructor, small animal clinic; Charles R. Wallace (API '56), research assistant, small animal medicine; A. M. Wiggins (API '46), after a year of graduate study, returned associate professor in charge of ambulatory clinic; F. G. Schell (API '41), associate professor, large animal surgery and medicine; J. T. Vaughan (API '55), instructor, ambulatory clinic; and H. C. Morgan (API '55), instructor, physiology.

The following resignations were reported: H. R. Seibold (UP '31), to accept a position as pathologist at Plum Island Animal Disease Research Station; R. E. Thorson, to accept a position with the research department, Lederle Laboratories, Pearl River, N.Y.; J. E. Neal, to take graduate work in bacteriology at Texas A. & M. College; and F. A. Clark (API '24),

TABLE 2—Veterinary Student Enrollment for the Academic Year 1956-1957

Schools	Fresh.	Soph.	Jun.	Sen.	Spec.	Grad.	Total 1956	Total 1955	Change
Alabama Polytechnic Institute	57	58	62	58	0	5	240	258	−18
California, University of	50	50	48	44	2	12	206	212	− 6
Colorado A. & M. College	66	71	63	61	0	6	267	265	+ 2
Cornell University	57	48	51	46	0	20	222	211	+11
Georgia, University of	63	58	64	53	1	0	239	241	− 2
Illinois, University of	50	27	30	33	1	16	157	137	+20
Iowa State College	71	71	60	55	1	11	269	278	− 9
Kansas State College	71	69	65	59	0	8	272	265	+ 7
Michigan State College	64	59	58	59	7	14	261	277	−16
Minnesota, University of	48	46	43	48	3	28	216	209	+ 7
Missouri, University of	30	30	29	29	1	5	124	122	+ 2
Ohio State University	72	72	70	69	0	18	301	298	+ 3
Oklahoma A. & M. College	42	39	35	32	2	7	157	143	+14
Pennsylvania, University of	56	47	46	45	0	0	194	201	− 7
Quebec Veterinary School*	27	29	23	18	0	0	97*	89*	+ 8
Texas A. & M. College	64	57	62	49	1	14	247	252	− 5
Toronto, University of*	54	51	52	45	4	7	213*	224*	−11
Tuskegee Institute	30	21	20	11	0	0	82	73	+ 9
Washington, State College of	51	47	48	38	0	10	194	185	+ 9
Totals	1,023	950	929	852	23	181	3,958	3,940	+18

*The Canadian schools give a five-year course instead of requiring two years of preprofessional collegiate credit. The first year enrollments (56 at Ontario and 35 at Quebec), therefore, are not included.

who taught veterinary public health and bacteriology, has retired.

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California.—Additions to the staff of the School of Veterinary Medicine at the University of California include: P. H. Allen (WSC '52), assistant specialist, parasitology; E. L. Biberstein (COR '51), microbiology; Julius Fabricant (UP '42), poultry pathology (on sabbatical leave from Cornell); A. C. Asbury (MSC '56), J. P. Hughes (KSC '56), Hyrum Kitchen (CAL '56), C. T. Robinson (CAL '56), G. H. Theilen (CAL '55), and Alida P. Wind (UT '53), medicine and surgery; L. Z. McFarland (CAL '56), anatomy; and H. L. Parker (CAL '52), radiobiology.

The following resigned to enter practice: B. L. Hutcherson (CAL '55), P. E. Kelley (COR '55), and B. J. Smith (CAL '55); and G. A. Linton (MSC '50), accepted another position.

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Colorado.—Dr. Rue Jensen, associate dean of veterinary medicine, School of Veterinary Medicine, Colorado A. & M. College, will become dean on Jan. 1, 1957, on the resignation of Dean Floyd Cross. Dr. L. A. Griner is on sabbatical leave at the University of California. New staff members include: Melvin J. Swenson (KSC '43), professor, physiology; Arch F. Alexander (MIN '51), instructor, pathology and bacteriology; Dale Angus (CAL '36), temporary instructor, veterinary clinics and surgery; John R. Collier (OSU '41), associate bacteriologist, experimental station; Sterling Dawley (ONT '49) and Bennie G. Erwin (COL '56), assistant professors, veterinary clinics and surgery; Cameron L. Seger (COL '53), temporary junior pathologist, experiment station; and Robert W. Thomassen, instructor, veterinary anatomy.

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Georgia.—Additions to the faculty at the School of Veterinary Medicine, University of Georgia, include: Helen Jordan (GA '55), pathology and parasitology; and Marvin L. Fleming (GA '55), anatomy and histology.

Richard E. Bradley (GA '54) and George W. Patterson (ALA '51) resigned to work toward more advanced degrees.

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Illinois.—Dr. Carl A. Brandly became dean of the College of Veterinary Medicine at the University of Illinois, Sept. 1, 1956, on the resignation of Dean Robert Graham. The following have joined the teaching staff: Richard J. Brown (ILL '56) and Albert O. Griffiths (ILL '56), veterinary clinical medicine; Paul S. Quinn (ILL '56), animal pathologist, State Department of Agriculture diagnostic laboratory cooperating with the College of Veterinary Medicine; and Julian P. Kreier (UP '53), instructor, veterinary physiology and pharmacology.

Harold S. Bryan (MSU '44) resigned to join the staff of Upjohn Co., Kalamazoo, Mich.

Iowa.—The following have been appointed to the faculty of the Division of Veterinary Medicine, Iowa State College: F. A. Anderson (ISC '32), assistant professor, medicine and surgery; W. E. Smith (COL '56), instructor, medicine and surgery; J. H. Bailey (ISC '46), assistant professor, medicine and surgery; J. Thomas Bell, Jr. (GA '52), assistant professor, veterinary anatomy; Charles E. Brandner (COL '56), instructor, veterinary hygiene; Wayne Kirkham (TEX '41), assistant professor, veterinary hygiene; and A. C. Strafuss (KSC '54), instructor, diagnostic laboratory.

The following have resigned from the staff: Eugene H. McGehee, Jr. (ISC '55), to enter practice; Robert Whiteford (GA '51), to study for the Ph.D. degree; L. E. Barnes (OSU '21), to join the staff of Eli Lilly and Co., Indianapolis, Ind.; T. F. Bartley, to accept a position with Corn States Serum Co., Cedar Rapids, Iowa; and Robert McClure (ISC '55) and J. R. Collier (OSU '41), to join faculties at other schools.

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Kansas.—New appointments to the faculty at the School of Veterinary Medicine, Kansas State College, are: Donald M. Trotter (KSC '46), anatomy; and Frank N. Dost (WSC '51), physiology.

Donald D. Johnson (TEX '44) and Wayne W. Kirkham (TEX '41) resigned from the Department of Pathology, and Dennis D. Goetsch (KSC '52) and Melvin J. Swenson (KSC '43), from the Department of Physiology.

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Michigan.—The following veterinarians have joined the staff of the College of Veterinary Medicine, Michigan State University: Lloyd C. Ferguson (OSU '34), head, Department of Microbiology and Public Health; Kenneth Whitehair (KSC '40), experiment station; C. C. Beck (MSU '54) and D. J. Ellis (MSU '43), ambulatory clinic; and Donald Piermattei (MSU '54), small animal clinic. Elliott Myers (MSU '55) resigned from the staff.

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Minnesota.—The faculty changes in the School of Veterinary Medicine, University of Minnesota, include the following appointments: Robert K. Anderson (COL '44), associate professor of veterinary bacteriology and public health; Lynn A. George (WSC '53), research fellow, veterinary pathology and parasitology; Richard C. Herschler (MIN '56), research fellow, veterinary obstetrics; Donald C. Innes (KSC '39), research fellow, veterinary surgery and radiology; Francis D. Knippling (MSU '50) and Joseph W. Walker (TEX '41), instructors, veterinary pathology and parasitology; and Al W. Stinson (GA '56), instructor, veterinary anatomy.

The following have resigned: Everett D. Besch (TEX '54), to accept a position at another school; Charles Gale (MSU '52), to join the staff at the agricultural experiment station,

Wooster, Ohio; and Donald R. Knauff (GA '54), to become research veterinarian for Hess and Clark Co., Ashland, Ohio.

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New York.—The following appointments have been made to the faculty of the New York State Veterinary College, Cornell University: J. Bentinck-Smith (COR '44), associate professor, recently returned from sabbatical leave in Sweden; Bruce Calnek (COR '55), assistant professor, poultry diseases; Richard S. Guthrie (COR '50) and Bernard F. Brennan (OVC '51), field veterinarians, mastitis program; J. M. King (OKL '55), instructor, pathology; Chintamini Singh (from India), research associate, virus laboratory; H. L. Bandemer (COR '55), resident veterinarian, small animal clinic; J. F. Kavanaugh (COR '54), resident surgeon; and John L. Hyde (COR '54), assistant professor, pathology. The new medical interns are E. E. Perona (COR '56) and Robert V. Manning (COR '55), small animal clinic; John Howe (COR '56) and M. P. Bartlett (COR '56), ambulatory clinic. The following faculty members will be on sabbatical leave for a year: M. E. Miller (COR '34), Julius Fabricant (UP '42), and Robert W. Dougherty (OSU '36) who went to New Zealand to study bloat in cattle.

Resignations include: D. T. Baker (COR '44), to go into practice; Edwin J. Hoff (COR '52), to join the staff of a commercial company; Lt. Col. Edwin J. Sunderville (COR '39), because of elimination of ROTC unit; Roger Panciera (OKL '53), to accept a position at Oklahoma A.&M. College; and Kyu Lee, to return to his home in Korea.

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Ohio.—The following additions have been made to the faculty of the College of Veterinary Medicine, Ohio State University: Edward F. Donovan (OSU '49), assistant professor, veterinary medicine; William E. Davis (OSU '56), instructor, veterinary medicine; B. B. Hancock (TEX '51), instructor, bacteriology; Robert Koestner (MUN '51), instructor, veterinary pathology; Philip W. Murdick (OSU '52), instructor, veterinary medicine; and Richard C. Piper (OCU '56), instructor, veterinary pathology.

The following have resigned: Earl J. Catcott (OSU '43), to engage in research; Lloyd C. Ferguson (OSU '34), to join the staff at Michigan State University; and George E. Maurice (COR '54) and James L. Gabel (OSU '55). On military leave are Albert A. Gabel (OSU '55), Richard A. Griesemer (OSU '53), and William J. Roenigk (OSU '54).

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Oklahoma.—Dr. Glenn C. Holm (ISC '36) became dean of the School of Veterinary Medicine, Oklahoma A. & M. College, Oct. 1, 1956. Other new appointments to the staff are: Andrew W. Monlux (ISC '42), professor and head of the pathology department; Everett D. Besch (TEX '54), assistant professor in parasitology; William J. Bracken (KSC '55) instructor in pathology; Ralph G. Buckner (KSC '56) and George P. Mayer (OKL '54), instructors in medicine and surgery; Kenneth W. Huffman (OKL '53), instructor in anatomy; Chester F. Meinecke (KSC '52), instructor in physiology and pharmacology; Roger J. Panciera (OKL '53), assistant professor in pathology.

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Texas.—New faculty members at the School of Veterinary Medicine, Texas A.&M. College, include: R. R. Bell (GA '52), associate professor, veterinary parasitology; Paul F. Jungerman (TEX '47), assistant professor, veterinary microbiology; J. N. Beasley (TEX '49), assistant professor, veterinary pathology; John P. Davis (TEX '53) and W. M. Romane (TEX '43), instructors, veterinary medicine and surgery; and R. T. Dubose (TEX '56) and J. R. Watkins (TEX '56), instructors, microbiology.

Resignations were accepted from E. M. Jordan (API '53), to enter practice; and E. E. Roth (TEX '48), to accept a position with Louisiana State University. James E. Neal (API '51) is on leave of absence for graduate study.

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Tuskegee.—Faculty changes at the School of Veterinary Medicine, Tuskegee Institute, include the following appointments: G. T. Blackledge (TUS '51), acting head, small animal clinic; Ellis M. Hall (TUS '55) instructor, small animal medicine; Harry T. Burleigh, Jr. (TUS '55), instructor, large animal medicine; and Raleigh H. Allen, Jr. (TUS '55), instructor, pathology and parasitology.

L. B. Mobiley (KSC '20) and J. H. White, Jr. (TUS '24), have resigned to enter practice.

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Washington.—The following appointments have been made in the faculty of the College of Veterinary Medicine at Washington State College: Ray E. Watts (COL '42), assistant professor, has returned from two years in Pakistan as a part of the exchange program between Washington State College and the University of the Punjab; Robert Leader (WSC '52), assistant professor, veterinary pathology, after a year of postgraduate study; and Dee Taylor (COL '54), instructor, ambulatory clinic.

Walton Hawk (COL '49) resigned to enter practice.

Dr. Marshak to Head Pennsylvania Department of Veterinary Medicine

Dr. Robert Marshak (COR '45), 33, has been appointed professor and chairman of the Department of Veterinary Medicine, School of Veterinary Medicine, University of Pennsylvania. He will assume his duties on Jan. 1,

1957, succeeding Dr. John D. Beck who has accepted a position with the New York Women's League for Animals.

Dr. Marshak, while maintaining a mixed practice (75% dairy cattle) for ten years at Springfield, Vt., conducted clinical investigations on parturient paresis, bovine ketosis, grass tetany, bovine bones, parathyroid activity, arsenic metabolism in cattle, toxicology of anti-

Dr. Robert Marshak

coccidials, ACTH and glucocorticoids, and also evaluated numerous new drugs. He is a member of the AVMA, American Association for the Advancement of Science, Vermont Veterinary Medical Association, American Dairy Science Association, and the New York Academy of Sciences; also of Phi Zeta and Phi Eta Sigma.

Dr. Marshak is the author and co-author of several scientific articles.

International Fertility Association Names Committee

The managing committee of the International Fertility Association has appointed an apposite committee on animal reproduction, nominating Dr. T. Bonadonna, Milan, Italy, as chairman. Drs. K. McEntee, Ithaca, N. Y., and N. Lagerlof, Stockholm, Sweden, have been named as members.

The object of the committee is to further the interest of veterinarians and biologists in the improvement of animal fertility and the struggle against sterility.

Dr. Ribelin Completes Research Fellowship, Appointed Professor of Pathology at Auburn

Dr. William E. Ribelin (ISC '49), on Sept. 30, 1956, received his Ph.D. degree from the University of Wisconsin where, for the past two years, he has been an AVMA Research Fellow. His research subject was the cytopathogenesis of vesicular stomatitis infection.

Dr. Ribelin was born in Oxnard, Calif., in 1924. He started his preveterinary training at Iowa State College in 1942 but because of three years of military (infantry) service, did not receive his D.V.M. degree until 1949. He accepted a position as instructor of pathology at Washington State

Dr. William E. Ribelin

College where he earned his M.S. degree in 1952. The results of his research there, on encephalitis in dogs, has been published (*Am. J. Vet. Res., 14,* (Jan., 1953): 96-104). He then spent two years in Amsterdam, Netherlands, as research veterinarian for the U.S.D.A., working on various aspects of foot-and-mouth disease, particularly the behavior of the virus in tissue culture (*Am. J. Vet. Res., 17,* (Jan., 1956): 40-44).

Dr. Ribelin is now on the staff of the School of Veterinary Medicine, Alabama Polytechnic Institute, where he accepted an appointment as a professor of pathology.

He is a member of the AVMA, the American College of Veterinary Pathologists, and the American Tissue Culture Association; also of Phi Kappa Phi, Sigma Xi, and Phi Zeta.

Dr. and Mrs. Ribelin have one son.

U. S. GOVERNMENT

Dr. Gooding Heads Disease Control Activities in California.—Dr. C. L. Gooding has been appointed veterinarian in charge of the U. S. Department of Agriculture's Animal Disease Eradication Branch and Animal Inspection and Quarantine Branch activities in Sacramento, Calif., effective Oct. 7, 1956. In his new assignment, Dr. Gooding is responsible for the administration of programs relating to the control and eradication of tuberculosis and brucellosis of cattle, scabies in sheep and cattle, bluetongue in sheep, and other major livestock diseases. He also has general supervision over all animal inspection and quarantine branch activities in the state.

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Dr. Nordstrom Named Veterinarian In Charge of ADE in Albuquerque.—Dr. E. H. Nordstrom (ISC '25) was appointed veterinarian in charge of animal disease eradication activities, U.S. Department of Agriculture, in

Albuquerque, N.M., effective Sept. 30, 1956. He replaces Dr. F. H. Sharp who recently retired.

Dr. Nordstrom was formerly stationed at Topeka, Kan., as assistant veterinarian in charge of animal disease eradication activities for the U.S.D.A. in Kansas, a post he had held since January, 1955.

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Veterinary Biological Inspectors Attend Refresher Course.—An intensive refresher and training course for U.S.D.A. veterinarians who inspect biological products was held at Michigan State University, East Lansing, Sept. 10-21, 1956.

The course was arranged by the Animal Inspection and Quarantine Branch of the Agricultural Research Service, U.S.D.A., and was conducted by the Department of Microbiology and Public Health of the University. Representatives of concerns manufacturing biological products, who helped in the planning of a similar course for the first time last year, participated directly in this year's lectures and laboratory work.

In attendance were 15 veterinarians engaged in the inspection of biological products at plants in seven states and in Washington, D. C. The course included a review of recent developments in bacteriology and virology and emphasized a study of techniques and equipment for manufacture and testing of the newer live virus and modified virus vaccines.

Instructors were: Drs. H. D. Anderson, Michigan Department of Health; C. H. Cunningham and J. P. Newman, Michigan State University; B. J. Gray, Fort Dodge Laboratories, Fort Dodge, Iowa; F. M. Murdock, Anchor Serum Co., South St. Joseph, Mo.; and A. L. Tellejohn, ARS, U.S.D.A., Washington, D. C.

AMONG THE STATES AND PROVINCES

Alberta

Southern Alberta Veterinarians Organize.—The Southern Alberta Veterinarians' Club held an organization meeting at the veterinary research laboratory, Lethbridge, on July 29, 1956. Drs. A. W. Allan, Cardston, and R. C. Connell, Lethbridge, were elected president and secretary-treasurer, respectively, and Dr. Donald MacLennan, Lethbridge, was elected chairman of the 1957 convention committee.

Following the meeting, a picnic was enjoyed by all attending veterinarians, their wives, and children.

s/R. C. CONNELL, *Secretary.*

Florida

State Association.—At the twenty-seventh annual meeting of the Florida State Veterinary Medical Association held at the San Carlos Hotel, Pensacola, Fla., on Sept. 23-25, 1956, the following officers were elected: A. E. Whaley, Kissimmee, president; E. Ben Plummer, Fort Meyers, president-elect; Robert P. Knowles, Miami, secretary; and Ralph W. Porter, Quincy, treasurer.

Officers of the Florida V.M.A. are (left to right)—A. E. Whaley, president; Ralph W. Porter, treasurer; and Robert P. Knowles, secretary.

s/ROBERT P. KNOWLES, *Secretary.*

Iowa

Eastern Iowa Association.—The forty-third annual meeting of the Eastern Iowa Veterinary Association was held at the Hotel Montrose, Cedar Rapids, on Oct. 4-5, 1956.

The following speakers and their subjects were included in the program: Joseph W. Albrecht, Princeton, Ill. (beef cattle practice); Robert Getty, Iowa State College, Ames (anatomy); Harlen Engelbrecht, Fort Dodge Laboratories, Fort Dodge, Iowa (swine diseases); Kenneth B. Haas, Loyola University, Chicago, Ill. (client relations); Richard E. Shope, M.D., New York, N. Y. (swine influenza and hog cholera); Richard A. Tjalma, University of Iowa, Iowa City (Iowa institute of agricultural medicine); Robert O. Sander, Waukon (dairy cattle practice). A panel discussion on swine by John W. Carey, West Liberty; James Ramsey, Tipton; Harlen Engelbrecht; Paul E. Maland, Charles City; and Joseph W. Albrecht was also included.

At the annual banquet and ball, Dr Shope spoke on "Some Observations Made on a Medical Mission to Russia."

s/F. E. BRUTSMAN, *Secretary.*

• • •

Urge that Veterinarian Head Sanitation Department.—Drs. E. G. Cole, R. A. Taussig, and H. C. Smith, all of Sioux City, represented the Siouxland Veterinary Medical Association before the city council, urging that a veterinarian be placed in charge of the city sanitation department. At present, veterinarians are hired on a part-time basis to inspect meat at three local packing companies and the present ordinance requires that a veterinarian accompany a health department official while investigating each dog bite case. The increased incidence of rabies in this area is a factor in requesting this change.—*Sioux City Journal-Tribune, Aug. 28, 1956.*

STATE BOARD EXAMINATIONS

Texas—The Texas State Board of Veterinary Medical Examiners announces that the next veterinary licensing examination will be held on Jan. 9-11, 1957, at Austin, Texas. The completed applications must be returned to the following address not later than 30 days before the examination date. Requests for applications and additional information should be addressed to: Mr. T. D. Weaver, executive secretary, Texas State Board of Veterinary Medical Examiners, 520 Littlefield Building, Austin 15, Texas.

VETERINARY MILITARY SERVICE

Air Force Veterinary Officers Awarded Graduate Degrees.—The Master of Public Health degree was awarded to the following career U. S. Air Force veterinary officers in June, 1956: Major Edward P. Hornickel, Capt. Donald C. Houk, Capt. William H. Grau, Jr., from the University of Michigan, East Lansing; Capt. Donald W. Ringley, University of California, Berkeley; and Capt. William V. Howells, Johns Hopkins University, Baltimore, Md. Captain Richard E. Benson received a Master's degree in medical physics and radiation biology from the University of Rochester, Rochester, N.Y.

• • •

Veterinary Officer Qualifies for Olympic Team.—Captain Adolph Weinacher, V.C., U.S.

West Virginia

Dr. Hershey Honored.—At the banquet held during the annual meeting of the West Virginia

Dr. Samuel Hershey (center) accepts the bronze plaque from Dr. V. H. Miller. Mrs. Hershey is on the left.

Veterinary Medical Association, Sept. 30-Oct. 1, 1956, at Parkersburg, a bronze plaque was presented to Dr. Samuel E. Hershey (QU '98), Charleston, "for his 63 active years of practice and contributions to the betterment of veterinary medicine in the State of West Virginia." Dr. Hershey is a charter member of the Association which was founded in 1897.

FOREIGN NEWS

Peruvian Veterinary Medical Association

A group of members of the Peruvian Veterinary Medical Association at a meeting, July, 1956, held to celebrate the election of Dr. Genaro Barragan as deputy to the Peruvian congress, the first veterinarian ever elected to the congress. In the front row, Dr. I. A. Merchant, dean, School of Veterinary Medicine, Iowa State College, Ames, is third from left. Dr. Barragan is fifth from right.

Air Force, won the National A.A.U. 50,000 meter walk at Baltimore, Md., on Sept. 16, 1956. He is presently training with the Olympic track and field team on the West Coast in

Captain Adolph Weinacher

preparation for the Olympic games to be held in Australia.

A native of Detroit, Mich., Captain Weinacher received his D.V.M. degree from Michigan State University, East Lansing, in 1951. He completed a tour of duty in the Far East and is now a base veterinarian at Willow Run Air Force Base, Belleville, Mich.

DEATHS

Jesse V. Bayles (TH '14), 74, Pennville, Ind., died on Aug. 12, 1956. Dr. Bayles had recently retired from practice.

C. N. Brookens, 77, Mount Pleasant, Mich., died Aug. 24, 1956. Dr. Brookens had been in ill health for several years. He is survived by his widow and two daughters, one son, six grandchildren, and two great grandchildren.

Rolland G. Brooks (IND '16), Farmland, Ind., died Aug. 27, 1956, from a heart attack. Dr. Brooks, a former state senator and representative, had served as a veterinarian for the Farmland area for 40 years. He was active in civic and professional activities and had served in World War I. His widow, two daughters, and five grandchildren survive.

★**Michael J. Cone** (ONT '07), 75, Pittsfield, Mass., died Aug. 22, 1956. Dr. Cone had served as a veterinary health officer in the Division of Livestock Disease Control for the state, having retired in 1951. He was a member of the Massachusetts State Veterinary Medical Association and of the AVMA. Dr. Cone is survived by his widow and a daughter.

John H. Fulstow (ONT '92), 95, Norwalk, Ohio, died Aug. 12, 1956. Dr. Fulstow was probably the oldest practicing veterinarian in Ohio. He had practiced in his community for 70 years. A native of England, Dr. Fulstow was an excellent horseman. He is survived by a son, a daughter, four grandchildren, and five great grandchildren.

Otto W. Haasis (GSN '13), 67, Lacombe, Alta., died Sept. 1, 1956, after a prolonged illness. Dr. Haasis is survived by his widow, a daughter, and a brother.

Benjamin C. Harrison (KCV '09), 89, Wichita, Kan., died May 11, 1955. Dr. Harrison worked in meat inspection and tick eradication with the BAI, U.S.D.A., from 1919 until 1920 when he joined the Kansas Live Stock Sanitary Commission where he remained until his retirement in 1947. He was a member of the Kansas and Oklahoma Veterinary Medical Associations.

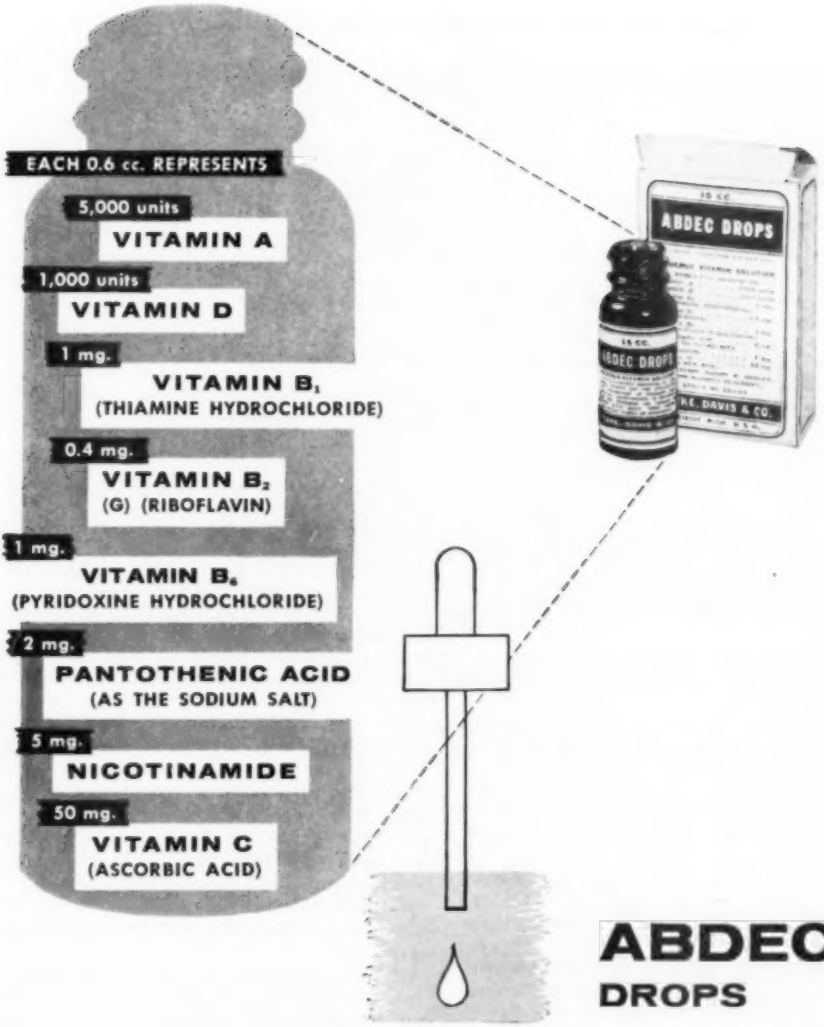
Wilbur R. Jackson (ONT '07), 78, Sanford, Maine, died Sept. 3, 1956. Dr. Jackson practiced in the Sanford area for 45 years and was a member of the Maine Veterinary Medical Association. He is survived by a son and a daughter.

Herbert L. Little (OSU '07), 70, Jeffersonville, Ohio, died Sept. 6, 1956, of a coronary occlusion. Dr. Little had practiced in Jeffersonville for 38 years. He retired in 1944. Dr. Little's survivors include his widow, two sons, three daughters, and seven grandchildren.

Ira W. McEachran (KCV '05), 80, Geneva, Neb., died Aug. 8, 1956. Dr. McEachran was interested in civic as well as professional activities and had served as a member and president of the Geneva city council for many years, as a member of the selective service board of Fillmore County during World War II, and as chairman of the county Republican committee for 20 years. He was past president of the Nebraska State Veterinary Medical Association and a member of the state Veterinary Examining and Licensing Board. Dr. McEachran is survived by his widow, a son, a daughter, and nine grandchildren.

Theodore C. Tiedebohl (CHI '07), 69, Coloma, Mich. died Aug. 18, 1956, after several months of ill health. Dr. Tiedebohl served, from 1906 to 1918, at the Chicago stockyards and was a cattle and horse inspector for the U. S. Government. He is survived by his widow, a daughter, and a granddaughter. He had been a member of the AVMA.

★Indicates members of the AVMA.



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Questions and Answers on Social Security

1) *Question* — Dr. Smith is married, reached age 74 in January, but continues to practice on a limited basis. He shows the following income on his 1956 tax return.

Net earnings from private practice	$3,000.00
Income on investments	1,800.00
Total adjusted gross income	$4,800.00

a) Does Dr. Smith pay Social Security tax? How much? When?

Answer — Dr. Smith, a veterinarian, would pay a self-employment tax of 3 per cent on the $3,000 net earnings from his private practice or $90 which is to be reported on his individual tax return for the year 1956.

For those who report on a calendar year basis, the return for 1956 is due on or before April 15, 1957.

b) When does Dr. Smith become eligible for benefits?

Answer — Dr. Smith who was 74 years old in January, 1956, can become eligible for benefits as soon as he has acquired six quarters of coverage. If he has no prior covered employment, he can be eligible for benefits as early as April, 1957.

c) Provided his income continues in 1957 on approximately the same level, will this income affect his benefits?

Answer — If his income in 1957 continues on approximately the same level, it will not affect receipt of Social Security benefits since he is past 72 years of age. The law provides for payment of benefits to any beneficiary 72 years old or older, regardless of the amount of their earnings.

d) Does the amount Dr. Smith earned from 1951 through 1955 have any bearing on the amount of his benefits after he becomes fully insured?

Answer — During the years 1951 through 1955, the self-employed veterinarian was not covered under the Social Security Act. The 1956 amendments to the Social Security Act provide for a five-year drop-out of low earnings or no earnings after 1950, which permits the Social Security Administration to disregard the years 1951 through 1955 in computing an individual's Social Security benefit.

2) *Question* — Dr. Jones spends most of his time in private practice, but he also works part-time on the state and federal Brucellosis Eradication Program.

His tax return for 1956 shows the following income:

Net earnings from private practice	$ 7,600.00
Salary for Brucellosis work	2,400.00
Total Adjusted gross income	$10,000.00

a) How much Social Security tax does Dr. Jones pay with his 1956 return?

Answer — The $2,400 salary which Dr. Jones, a veterinarian, receives in 1956 for his work on the state and federal Brucellosis Eradication Program is wages from employment covered under the Social Security Act, since he is classified as a part-time federal employee. Dr. Jones would pay a self-employment tax for 1956 of 3 per cent on $1,800, or $54, which is the difference between the $4,200 maximum combined wages and self-employment income and the $2,400 salary.

AVMA Adopts Health and Accident Insurance Plan

(AVMA REPORT—*Continued from adv. p. 10*)

fit is payable for accidental death, dismemberment, or loss of USE of both hands.

The amount of principal sum for each insured member is determined by the amount of his monthly income insurance, and a percentage of the principal sum is payable for each accidental loss.

Completing the AVMA Program is an optional "Catastrophe Hospital and Surgical Plan" for insured members up to age 65 and their dependents. The benefits are: $14.00 per day for hospital room and board up to 180 days, compared to the usual maximum of around 70 days; 100 per cent of all other hospital charges between $50 and $450, plus 75 per cent of all such charges thereafter up to $2,500; a schedule of surgical benefits up to $200.

Maximum benefits under these coverages would total $4,657.50, and the full amount is available for each separate illness or accident. There is no cumulative maximum.

Maternity benefits are payable with or without hospitalization after a dependent has been insured nine months.

All who apply will be insured regardless of physical condition, provided the quota for the basic monthly income is fulfilled and at least half of those who enroll also apply for the hospital and surgical insurance.

New Approach to Premium Structure

A distinct feature of the AVMA plan is that a member is offered the insurance at a premium determined by his age at the time he enrolls, which does *not* increase when the member reaches the next age bracket. The premium remains the same throughout the period of his participation in the plan.

Cooperation with State Programs

The Committee on Insurance gave a great deal

(*Continued on p. 28*)

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(INSURANCE PLAN—*continued from p. 26*)

of thought to the questions of developing a program that would not discommode members and groups enrolled in various state association plans. It was the recommendation of the Committee that the plan will be offered in all states except those requesting through an official communication from the state association that the AVMA plan *not* be offered to the AVMA members in their state. In addition, the AVMA program provides that full benefits will be paid under the AVMA plan, regardless of any other insurance a member has.

Mutual Life Insurance Company of New York will be the underwriters of the plan. Mr. Mitchell Hutchinson, Professional Society Group Insurance Company, will administer the program. All inquiries regarding the new AVMA Group Insurance Program should be directed to the

AVMA Group Insurance Trust,
Mitchell Hutchinson, C.L.U., Administrator,
Professional Society Group Insurance Co.,
120 South LaSalle St.,
Chicago 3, Ill.

Premium for Liability Insurance Increased

On adv. page 4 of this issue is announcement of an increase, effective Oct. 1, 1956 (in all but 8 states), in the premium for professional liability insurance available to AVMA members. This increase was promulgated by the National Bureau of Casualty Underwriters (the official rule and rate-making body) as the result of a review of the loss experience in such insurance in recent years.

The state insurance departments of Iowa, Louisiana, New York, North Carolina, South Dakota, Tennessee, Virginia, and Washington have not yet approved the increase. Hence, AVMA members in those states may take out or renew liability coverage at the old rates until a change is approved.

Information leaflets and rate schedules may be obtained from the AVMA, 600 S. Michigan Ave., Chicago 5, Ill.

WOMEN'S AUXILIARY

President—Mrs. A. E. Coombs, Box 174, Skowhegan, Maine
Secretary—Mrs. F. R. Booth, 3920 E. Jackson Blvd., Elkhart, Ind.

Florida Auxiliary.—At the ninth annual meeting of the Women's Auxiliary to the Florida State Veterinary Medical Association held Sept. 23-25, 1956, at the San Carlos Hotel, Pensacola, in conjunction with the twenty-seventh annual meeting of the Florida State Veterinary Medical Association, the following officers were elected for the ensuing term: Mrs. Ronald F. Jackson, St. Augustine, president; Mrs. George Lewallen, St. Petersburg, first vice-president; Mrs. J. D. Martin, Live Oak, second vice-president; Mrs. George Yopp, Jacksonville, secretary; Mrs. Peter S. Roy, Jacksonville, treasurer; and Mrs. William B. Martin, Gainesville, member-at-large.

The committee reported that auxiliary funds have been sent to the University of Georgia School of Veterinary Medicine, Alabama Polytechnic Institute School of Veterinary Medicine, AVMA Research Fund, and the Dr. J. V. Knapp Memorial Fund.

s/Mrs. George Yopp, *Secretary.*

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Eastern Iowa Auxiliary.—At the October 4 meeting of the Women's Auxiliary to the Eastern Iowa Veterinary Association, held in conjunction with the forty-third annual meeting of the Eastern Iowa V.M.A. at the Hotel Montrose, Cedar Rapids, the following officers were elected for the ensuing term: Mrs. F. E. Brutsman, Traer, president; Mrs. J. M. Barclay, Brooklyn, president-elect; Mrs. A. J. McIntosh, La Porte City, vice-president; Mrs. Leroy H. Bell, Davenport, secretary; and Mrs. K. H. Randolph, Lost Nation, treasurer.

s/Mrs. Leroy H. Bell, *Secretary.*

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Officers of the Women's Auxiliary to the Florida State Veterinary Medical Association are (left to right)—Mrs. George Yopp, secretary; Mrs. Ronald Jackson, president; and Mrs. Peter S. Roy, treasurer.

APPLICATIONS

Applicants—Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent associations shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. This certification was made by the secretary of the constituent association in accordance with Section 2, Article X, of the Administrative Bylaws.

BEASLEY, HUGH C.
861 N. Leak St., Southern Pines, N. Car.
D.V.M., Alabama Polytechnic Institute, 1947.

BECTON, PAUL
1848 Stanford Ave., Baton Rouge, La.
D.V.M., Alabama Polytechnic Institute, 1945.

BERGLUND, DAVID C.
1400 Roosevelt Rd., Hillside, Ill.
D.V.M., Ohio State University, 1949.

GRAY, STANLEY E.
121 3rd Ave., S.W., Aberdeen, S. Dak.
D.V.M., Ontario Veterinary College, 1939.

JONES, EARL M.
4033 Mescalero Rd., N. E., Albuquerque, N. M.
D.V.M., Alabama Polytechnic Institute, 1951.

McCORMACK, BIRTWHISTLE
214 Brush Creek Blvd., Kansas City, Mo.
D.V.M., Kansas State College, 1912.

PALMS, ANDREW G.
900 Burke Ave., Harvey, N. Dak.
D.V.M., University of Latvia, Riga, 1937.

PHELPS, GUY J., Jr.
3129 Atlanta Hgwy., Montgomery, Ala.
D.V.M., Alabama Polytechnic Institute, 1946.

SIMS, FRANK H.
3402 Main, Vernon, Texas.
D.V.M., Texas A. & M. College, 1951.

SPIERS, P. M.
Sandersville, Ga.
D.V.M., University of Georgia, 1928.

Applicants—Not Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and the names of his endorsers.

First Listing

MORAN, GUY G.
822 Rigsby, San Antonio, Texas.
D.V.M., Texas A. & M. College, 1945.
Vouchers: A. L. Hogge, Jr., and W. J. Schneider.

REYES, JULIO
Col. Amer. E-17, N. Rosita, Coahvila, Mexico.
D.V.M., Texas A. & M. College, 1955.
Vouchers: G. S. Trevino and R. J. Beamer.

SCHROEDER, GERALD F.
Box 187, Amenia, N. Y.
D.V.M., Colorado A. & M. College, 1942.
Vouchers: W. W. Rosser and W. E. Jennings.

UPHAM, ROY W.
9927th TU MDHS., 1819 W. Pershing Rd., Chicago, Ill.
D.V.M., Kansas State College, 1943.
Vouchers: G. W. Vacura and J. B. Young.

ZIEROLD, PABLO
Apdo Postal 32, Tlalnepantla, Mexico.
D.V.M., National School of Veterinary Medicine and Zootechnics, San Jacinto, Mexico, 1939.
Vouchers: A. H. Alexander and J. G. Hardenbergh.

Graduate Applicants

The following are graduates who have recently received their veterinary degree and who have applied for AVMA membership under the provision granted in the Administrative Bylaws to members in good standing of student chapters. Applications from this year's senior classes not received in time for listing this month will appear in later issues. An asterisk (*) after the name of a school indicates that all of this year's graduates have made application for membership.

First Listing

University of California

CONNOLLY, ROBERT J., D.V.M.
243 Roan Dr., Eugene, Ore.
Vouchers: W. M. Swangard and A. C. Baronti.

Colorado A. & M. College

HAWLEY, JOHN F., D.V.M.
P.O. Box 769, Chadron, Neb.
Vouchers: W. A. Aanes and B. O. Woodworth.

KEISS, CHARLES W., D.V.M.
Rt. 3, Grand Island, Neb.
Vouchers: G. W. Vacura and J. B. Young.

Ontario Veterinary College

MOSZKOWSKI, VICKI, D.V.M.
2172 McKenzie Pl., Niagara Falls, Ont.
Vouchers: J. Archibald and J. Henderson.

Second Listing

Colorado A. & M. College

OLSEN, DONALD B., D.V.M., 93 W. 2nd North, Smithfield, Utah.

Michigan State University

HARRIS, ROBERT E., D.V.M., 939 Main St., Crete, Ill.
PLYMALE, HARRY H., D.V.M., 11838 Allard St., Norwalk, Calif.

University of Missouri

PRICE, BILL A., D.V.M., 198 E. Chicago, Marcline, Mo.

University of Pennsylvania

MALLINSON, EDWARD T., V.M.D., c/o Dr. T. W. Brown, Fawn Grove, Pa.
WAMPLER, DONALD, V.M.D., 121 Ridge Rd., Moses Cake, Wash.

Texas A. & M. College

PAYNE, BOBBY J., D.V.M., Rt. 4, Box 39, West Monroe, La.

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COMING MEETINGS

Arizona Veterinary Medical Association. Annual meeting. Arizona Inn, 2200 East Elm St., Tucson, Dec. 3-4, 1956. John L. Hinds, 22 E. Ventura St., Tuscon, secretary.

Nebraska Veterinary Medical Association. Annual meeting. Hotel Lincoln, Lincoln, Dec. 3-5, 1956. W. T. Spencer, 1250 North 37th St., Lincoln, secretary.

Kentucky Veterinary Medical Association. Winter meeting. Phoenix Hotel, Lexington, Dec. 10-11, 1956. Robert H. Singer, 136 Shawnee Pl. Lexington, secretary.

Armed Forces Institute of Pathology. Postgraduate short course, pathology of diseases of laboratory animals. Armed Forces Institute of Pathology, Walter Reed Army Hospital, Washington, D. C., Dec. 10-14, 1956. The Director, Armed Forces Institute of Pathology, Walter Reed Army Medical Center, 6825 16th Street, N.W., Washington 25, D.C.

American Association of Equine Practitioners. Annual meeting. LaSalle Hotel, Chicago, Ill., Dec. 16-18, 1956. T. E. Dunkin, 1736 E. 71st Pl., Chicago 49, Ill., secretary.

New York State Veterinary College. Annual conference for veterinarians. New York State Veterinary College, Cornell University, Ithaca, Jan. 2-4, 1957, W. A. Hagan, dean.

Pennsylvania, University of. Annual conference for veterinarians. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa., Jan. 8-9, 1957. M. W. Allam, dean.

Tennessee Veterinary Medical Association. Annual meeting. Memphis, Tenn., Jan. 13-15, 1957. H. W. Hayes, 5009 Clinton Pike, Knoxville, secretary.

Oklahoma Veterinary Medical Association. Annual meeting. Huckins Hotel, Oklahoma City, Jan. 13-15, 1957. M. N. Riemenschneider, 122 State Capitol Building, Oklahoma City, secretary.

Michigan State University. Annual postgraduate conference for veterinarians. College of Veterinary Medicine, Michigan State University, East Lansing, Jan. 22-23, 1957. C. F. Clark, dean.

Iowa Veterinary Medical Association. Annual meeting. Hotel Fort Des Moines, Iowa, Jan. 22-24, 1957. F. B. Young, Waukee, Iowa, secretary.

Maryland State Veterinary Medical Association. Winter meeting. Lord Baltimore Hotel, Baltimore, Jan. 23-24, 1957. John D. Gadd, Cockeysville, Md., secretary.

Intermountain Veterinary Medical Association. Twenty-ninth annual meeting. Hotel Utah, Salt Lake City, Utah, Jan. 24-26, 1957. R. A. Bagley, 2387 East 39th South St., Salt Lake City, Utah, secretary.

Virginia Veterinary Medical Association. Annual meeting. Hotel John Marshall, Richmond, Va., Jan. 27-29, 1957. W. B. Bell, 1303 Hillcrest Dr., Blacksburg, secretary.

California State Veterinary Medical Association. Midwinter conference. School of Veterinary Medicine, University of California, Davis, Jan. 28-30, 1957. Charles S. Travers, 3004 16th St., San Francisco, executive secretary.

Louisiana State University. Conference for veterinarians. Pleasant Hall, Louisiana State University, Baton Rouge, Jan. 29-30, 1957. W. T. Oglesby, head, Department of Veterinary Science.

Minnesota State Veterinary Medical Association. Annual meeting. Radisson Hotel, Minneapolis, Feb. 4-6, 1957. B. S. Pomeroy, 1443 Raymond Ave., St. Paul 8, secretary.

New Jersey, Veterinary Medical Association of. Annual meeting. Berkeley Carteret Hotel, Asbury Park, Feb. 13-14, 1957. J. R. Porteus, P. O. Box 938, Trenton 5, N. J., resident secretary.

Illinois State Veterinary Medical Association. Annual meeting. LaSalle Hotel, Chicago, Feb. 26-27, 1957. C. B. Hostetler, 1385 Whitcomb Ave., Des Plaines, secretary.

(*Continued on p. 32*)

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REFERENCES: 1. Blount, W. P.: Vet. Rec. 67:1087 (Dec. 10) 1955. 2. Pote, D. D.: Southeast Vet. 6:22 (Winter) 1955. 3. Smith, H. W.: J. Comp. Path. 65:55 (Jan.) 1955. 4. Titkemeyer, C. W., and Schmittle, S. C.: Abstracts 45th Ann. Meeting Poultry Sc. Ass., Raleigh, N. C., Aug. 7-10, 1956, p. 49.

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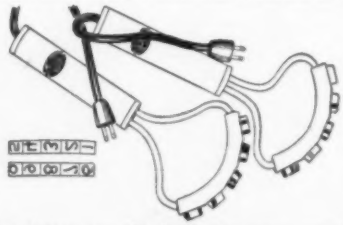

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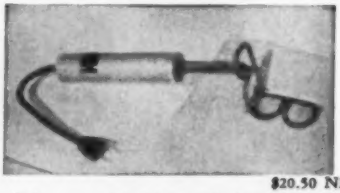

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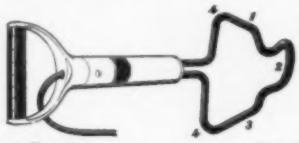

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(COMING MEETINGS—*continued from p. 30*)

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the first Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2, Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda Contra Costa Veterinary Medical Association, last Wednesday of each month. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of each month. E. Paul, Redwood City, Calif., secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. T. D. Harris, San Mateo, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

(*Continued on p. 34*)

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COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the second Friday of each month, time and place specified monthly. James B. Murphy, Eustis, Fla., secretary.

Jacksonville Veterinary Medical Association, the second Thursday of each month, time and place specified monthly. George F. Yopp, 4644 Main St., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. Harold A. Tennant, Atmore, Ala., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. Ross E. Evans, 5215 S. Dixie Highway, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Tuesday of each month, at the Seven Seas Restaurant, Miami, Fla. E. D. Stoddard, 6432 S. W. 8th St., Miami, Fla., secretary.

Suwannee Valley Veterinary Association, the third Friday of each month, at the Thomas Hotel, Gainesville, Fla. R. C. Mann, Rt. 1, Box 37, Ocala, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association the third Thursday of each month. W. E. Sharp, Union City, Ind., secretary.

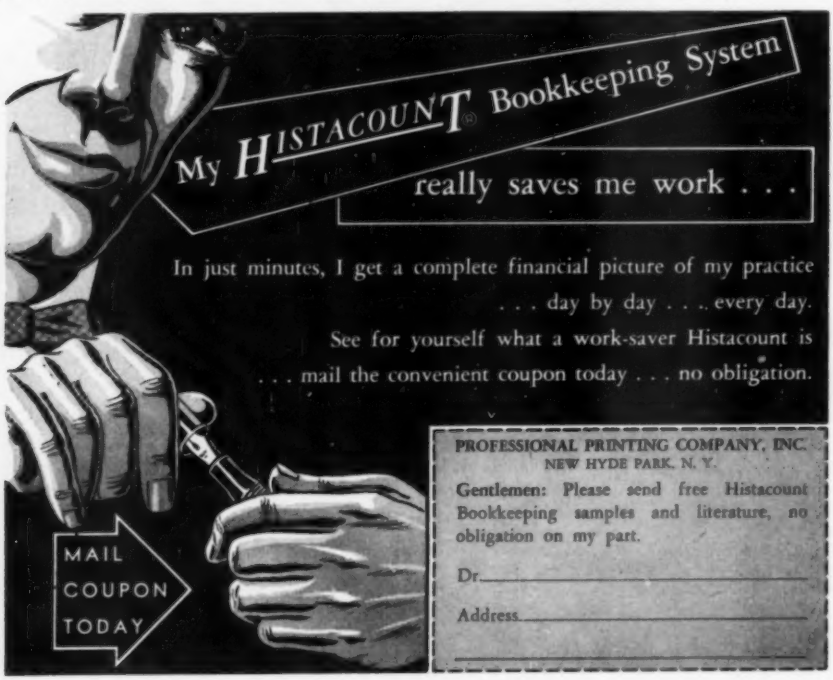
IOWA—Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. H. V. Henderson, Reinbeck, Iowa, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

(*Continued on p. 36*)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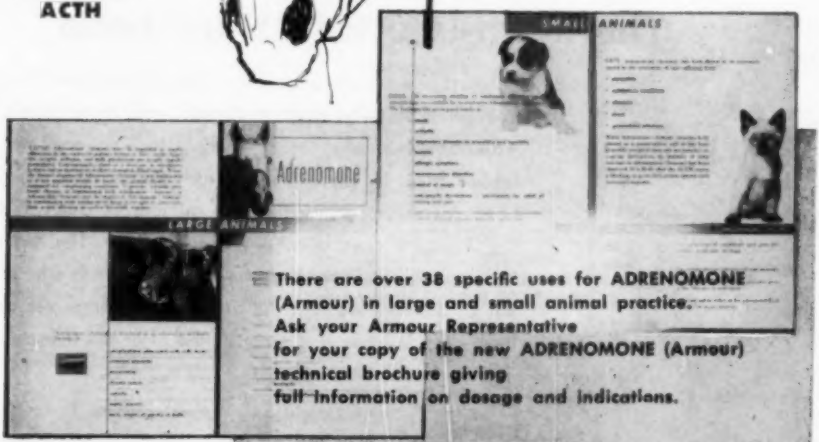

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(COMING MEETINGS—*continued from p. 34*)

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankfort, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of the month (except July and August) at the Sheraton Hotel, Spring Ave. and Lindell Blvd. Allen B. Shopmaker, 136 N. Meramec, Clayton 5, Mo., secretary.

Kansas City Small Animal Hospital Association, the first Monday of each month, at alternating hospitals. W. F. Noland, 7504 Metcalf, Overland Park, Kan., secretary.

Kansas City Veterinary Medical Association, the third Tuesday of each month at Exchange Hall, ninth floor, Livestock Exchange Bldg., 1600 Genessee St., Kansas City, Mo. Busch Meredith, 800 Woodswether Rd., Kansas City 5, Mo., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Highstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. F. B. Duke, 49 Taylor St., High Bridge, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month. Wm. Allen Potts, 401 W. James St., Mount Olive, secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. John G. Martin, Boone, N. Car., secretary.

(*Continued on p. 38*)

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(COMING MEETINGS—*continued from p. 36*)

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month. James M. Brown, 2818 W. Britton Rd., Oklahoma City, secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary St., Richmond 20, Va., secretary.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, Va., secretary.

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Tuesday of each month in the Trinity Episcopal Church, 8th and James St., Seattle, Wash. P. R. Des Rosiers, 5508 2nd Ave., N. W., Seattle 7, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. L. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. George F. Lynch, 201 West Devon St., Milwaukee 17, Wis., secretary.

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Veterinary assistant wanted in small animal practice. Address Davis Animal Hospital, West Main Street, Stamford, Conn.

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Prominent pharmaceutical manufacturer requires a veterinary salesman in its foreign operation; must be fluent in Spanish. Considerable travel throughout Latin America; D.V.M. degree desirable. Address "Box Y 1," c/o JOURNAL of the AVMA.

Veterinarian wanted for staff of large hospital for small animals in New York City. Address "Box Y 2," c/o JOURNAL of the AVMA.

Veterinarians wanted for disease control program. For further information contact Dr. K. J. Peterson, State Veterinarian, State Department of Agriculture, Salem, Ore.

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(Continued on p. 44)

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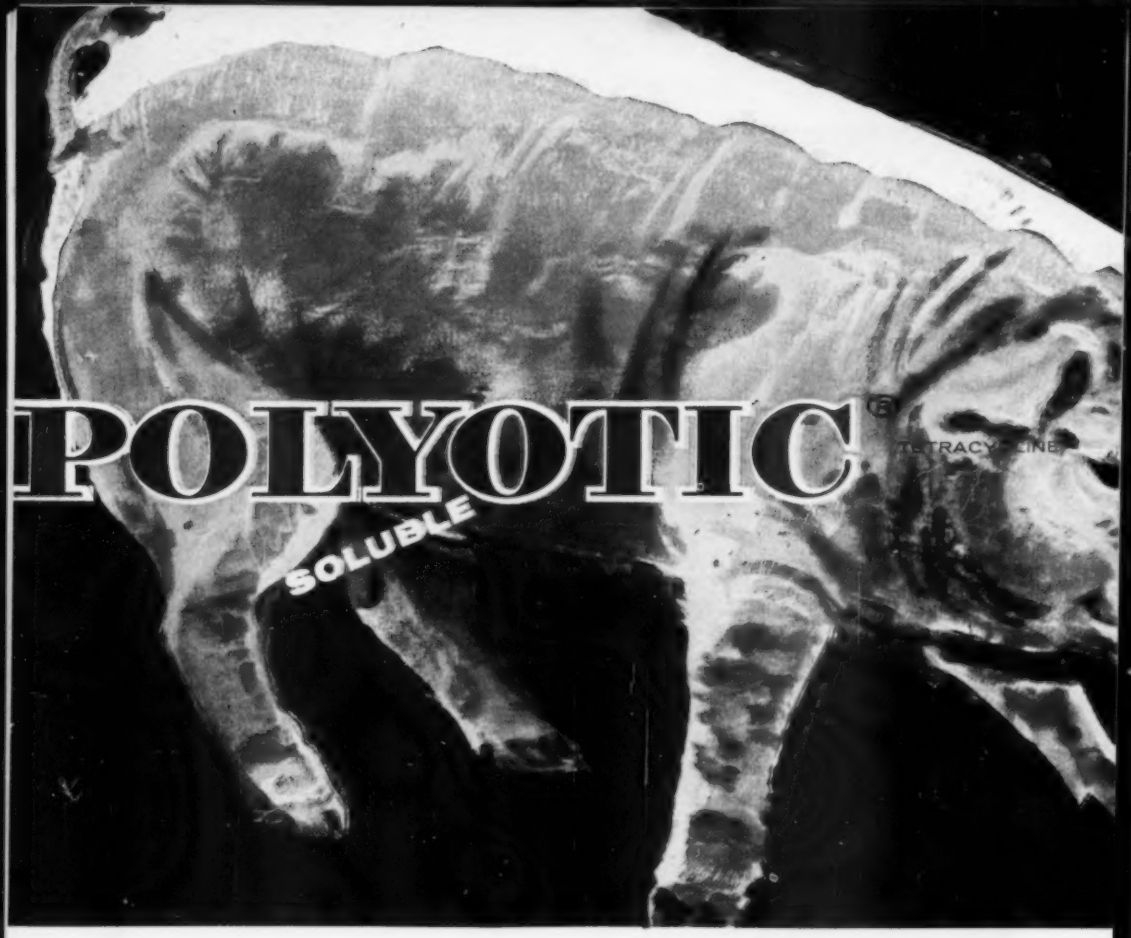

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(CLASSIFIED ADS—*continued from p. 40*)

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West Coast veterinary pharmaceutical manufacturer desires graduate veterinarian, 25 to 35 years old, for active working position in production leading to department head status. California license not required. Address "Box Y 15," c/o JOURNAL of the AVMA.

Wanted—Positions

Woman veterinarian, recent graduate, desires position with small animal practitioner in East; licensed in New York and Ohio. Address "Box Y 3," c/o JOURNAL of the AVMA.

Recent Cornell graduate desires position in small animal practice, licensed in New York, Pennsylvania, Virginia. Being discharged from army, December, 1956. Married. Address "Box Y 12," c/o JOURNAL of the AVMA.

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Young veterinarian desires position in pharmaceutical or serum company. Experienced in research, and with large and small animals; good clinician, surgeon, pathologist. Married. Address "Box Y 14," c/o JOURNAL of the AVMA.

Extension or teaching position wanted by general practitioner; mature in experience, young enough for new idea. Address "Box Y 16," c/o JOURNAL of the AVMA.

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Large 6-room house; excellent condition, on busy 4-lane highway, Scarsdale, N.Y. Used 30 years as a boarding kennel; excellent site for a veterinarian. For rent at $275 per month. Address D. Sapey, 636 Central Ave., Scarsdale, N.Y.

For Sale or Lease—Practices

Small animal clinic for sale in large Texas city. Bargain price, $22,000, includes real estate and practice; $5,000 down plus drugs and equipment. Address "Box X 15," c/o JOURNAL of the AVMA.

Pet hospital for sale, East Bay, northern California; $5,000 buys practice, equipment, drugs and supplies, furnishings, neon sign. Buyer gets 7-year lease on hospital at $100 monthly; gross, $700-$1,000 monthly, potential higher. High fees, short hours, climate excellent. No ulcer producer. Address "Box Y 9," c/o JOURNAL of the AVMA.

(*Continued on p. 45*)

IN CNS EMERGENCIES . . .

These Proven Stimulants

are used successfully to stimulate the Central Nervous System in narcotic poisoning, respiratory and circulatory collapse, and in physical exhaustion.

STIMULINE

Nikethamide, 25% solution for intravenous, intramuscular, or oral use.

ALSO INDICATED IN: shock, hemorrhage, septicemia, and pneumonia. May be used with pentobarbital anesthetics for more complete anesthesia with minimum effect on respiration and circulation.

	1	6	
30cc. vial	$ 1.00	$ 5.40	
125cc. vial	$ 3.50	$18.90	
	12	36	72
30cc. vial	$10.00	$28.50	$ 54.00
125cc. vial	$35.00	$99.75	$189.00

AMPHETAMINE SULFATE

Amphetamine Sulfate racemic, 50 mg. per cc.

ALSO INDICATED IN: parturient paresis, azoturia, heat stroke, lack of libido in sires and other conditions requiring stimulation of the vital functions.

	1	6	12
30cc. vial	$ 1.75	$ 9.45	$ 17.50
	36	72	144
30cc. vial	$49.87	$94.50	$175.00

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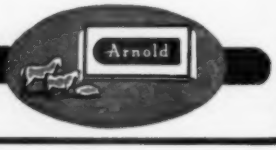
NEW CASTLE, INDIANA



(CLASSIFIED ADS—*continued from p. 44*)

Small animal practice in Coral Gables, Fla., for lease at $4,800 annually to veterinarian with Florida license. Address Brown, 4 East 77 St., Apt. 5, New York, N.Y. Telephone. LE 5-0056.

Mixed practice for sale in Maryland on good highway; 80% large animals, 20% small; fine dairy practice established. Home with full basement included. Address "Box Y 4," c/o JOURNAL of the AVMA.

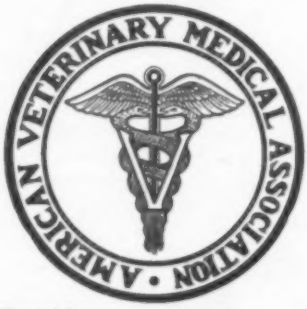
Mixed practice with well-equipped pet hospital for sale; on double lot facing main highway in Middlewest. Address "Box Y 7," c/o JOURNAL of the AVMA.

Mixed practice for sale, southeast Texas; serves entire county; county seat of approximately 20,000. Hospital and 1 acre of property, $10,000. Address Dr. W. S. Nichols, Box 342, Woodville, Texas.

Good practice for lease in Southeast; equipment, office, residence—only need car. Mostly large animals; good potential, small animals. Test program bringing $2,000; other inspection work, $2,500-$3,000. Owner to be gone 2 years. Reasonable lease; option to right man. Address "Box Y 10," c/o JOURNAL of the AVMA.

Mixed practice, mostly small animals, hospital, and equipment for sale: established for years in growing section of mid-Florida. Fair amount of capital will handle. Reason, other interests. Address "Box Y 11," c/o JOURNAL of the AVMA.

(*Continued on p. 46*)



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Your Symbol of Prestige

- AUTO EMBLEMS

Solid metal and weather-resistant. Attaches to license plate or license-holder. 3½ inches in diameter. Price $2.50 each.

- DECALS

Easy-to-apply on office doors or windows. 3½ inches in diameter. Price $.25 each.

- LAPEL PINS

Satin-finish surface featuring the AVMA emblem. Popular screw-post design. Price $.60 each.

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Please send, postage paid, the membership items checked:

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( ) Payment enclosed

( ) Bill me

Name .....
(Please print)

Street .....

City ..... State .....

(CLASSIFIED ADS—*continued from p. 45*)

Miscellaneous

Pregnancy diagnosis in mares—45th to 150th day. Request mailing tubes; $7.00, 2 or more, $6.00 each. Pregnancy Diagnostic Laboratories, Dysart, Iowa.

Breedersleve—the disposable obstetrical sleeve. Package of 25 with detachable chest band, $5.00; lower wholesale prices. Free sample upon request. Breeders Equipment Co., Flourtown, Pa.

Artificial Udder—Beco Litter Feeder—nurses 8 puppies simultaneously. Stays warm; easily cleaned and sterilized. Complete unit, $15; guaranteed. Breeders Equipment Co., Flourtown, Pa.

We wish to locate the mother of a huge male boxer by the name "Ronnie." $25 reward if she is located. Address J.R.H., Box 2349, Knoxville, Tenn., or call Knoxville 5-0241 and ask for Jim.

A young elk, which preferred ranch headquarters to the wilds, was awarded the title of "most sanitary elk of 1955." He liked the dipping tank so well that he "managed to lead each lot (of calves) into the tank."—*Farm J., Sept., 1956.*

Unique "Grandfather" Photo Contest

If you have or can produce a good photograph of a grandparent-grandchild combination among your dogs, you would do well to enter it in a unique contest just announced by the Gaines Homogenized Dog Meal people.

Forty-two prizes totalling $1,500 in cash are offered in the contest for good, interesting pictures of a dog or bitch with one of his or her grandsons or granddaughters, or even a great-grandchild. Pictures of purebreds only are eligible, but the grandchild must be at least 1 year old. The pictures can be in black and white or color, and one person can enter as many different ones as he likes.

Pertinent data should be on the back of each of the pictures, which should be mailed to Box 103, Dept. 2, Kankakee, Ill. Deadline for contest entries is midnight, March 31, 1957.

The Nature *of* Brucellosis

BY WESLEY W. SPINK, M. D.

The product of twenty years of work with human brucellosis at the University of Minnesota Hospitals is presented in this thorough survey, with detailed descriptions of methods of diagnosis and treatment. There is also a comprehensive view of animal brucellosis and a report on new work in basic research on the disease. Over 60 illustrations and a bibliography of almost 1000 references are included. $8.00

At your bookstore, or from
The University of Minnesota Press,
Minneapolis 14, Minn.

Cancer can't strike me, I'm hiding.

Cancer?

The American Cancer Society says that too many people die of it, NEEDLESSLY! That's why I have an annual medical checkup however well I feel. I know the seven danger signals. And when I want sound information, I get it from my Unit of the

AMERICAN CANCER SOCIETY

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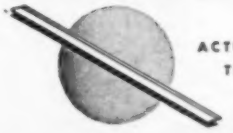
Compare RESULTS

and you will always Prescribe MASTICS®



ACTUAL SIZE
THE ORIGINAL
UDDER BOUGIE

MASTICS P&S

100,000 units penicillin
50,000 mcg. dihydrostreptomycin

MASTICS act fast because medication in high concentration is quickly dispersed throughout the quarter. Improvement often noted in 12 hours.

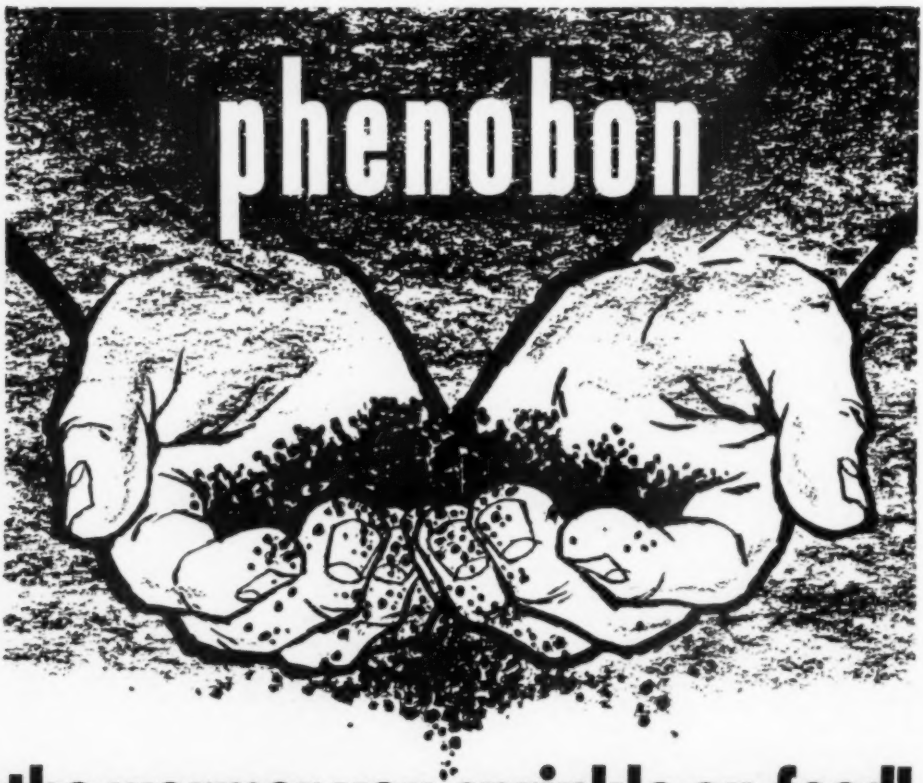
MASTICS contain no grease, no wax, no insoluble materials to remain in the udder retarding antibiotic action. MASTICS milk out completely—produce no residue on the strainer.

MASTICS are so effective, cows are returned to the herd more promptly with less loss of production.

LOW IN COST...HIGH IN POTENCY
MASTICS SAVE TIME, MONEY, MILK

WRITE FOR SAMPLES AND PRICES

The Martin Laboratories
West Chester, Penna.



the wormer you sprinkle on feed!

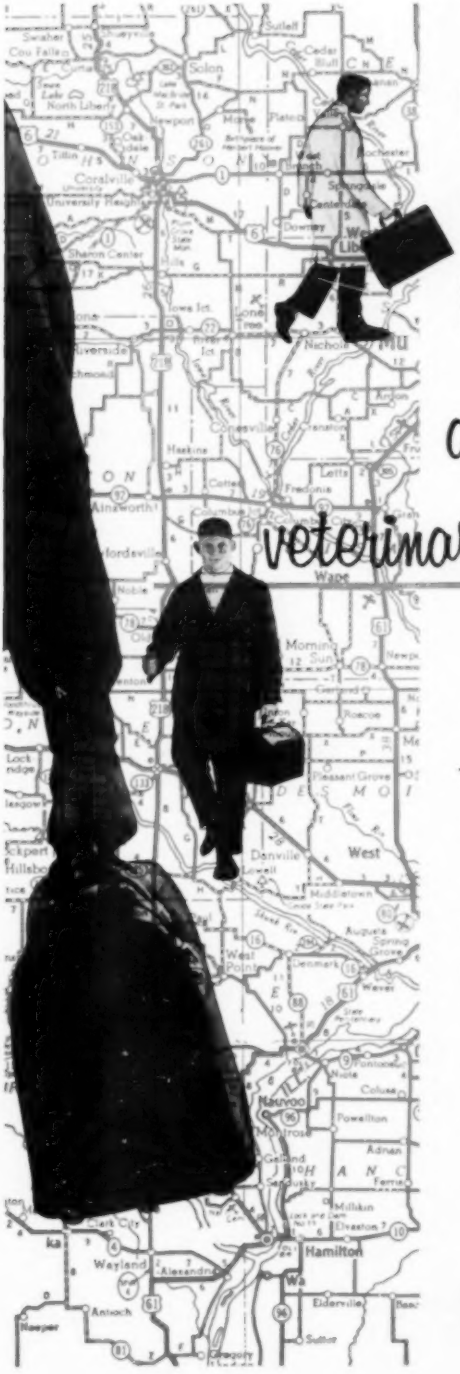
Phenobon is a highly effective anthelmintic for the control of hookworms, nodular worms, stomach worms, and palisade worms in sheep; strongyles in horses; cecal worms in poultry. It is safe for treating young or unthrifty animals.

With Phenobon your clients treat any number of animals quickly and easily by sprinkling Phenobon on their feed. Animals actually like the taste of Phenobon and seek out the granules.

Phenobon contains phenothiazine and carob flour, producing a pleasant tasting preparation. Carob flour slows the passage of the phenothiazine through the intestinal tract by clinging to the intestinal wall of the host and the cuticle of the parasite. This prolongs the vermicidal action.

Because of its effectiveness and convenience, Phenobon is an excellent dispensing item.

HAVER-GLOVER *Laboratories* INCORPORATED



Lockhart's Ovine Origin Rabies Vaccine...

a favorite of veterinarians everywhere!

This vaccine is a sterile, neutral, phenolized 20% suspension prepared from the brain and spinal cord of sheep showing typical symptoms of rabies following subdural injections of the fixed virus of rabies. Veterinarians everywhere have found it gives excellent results.

Lockhart also offers the graduate veterinarian modified live virus rabies vaccine of chick embryo origin. Protect more patients with Lockhart rabies vaccines.

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HAVER-LOCKHART LABORATORIES
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Sales to graduate veterinarians only.

it takes all three to make a superior RABIES VACCINE

COMMERCIAL VACCINE COMPARISON

product ▼	1 tissue concentration	2 particle size	3 reconstitution
A	9%	large	very poor
B	10%	medium	very poor
C	7%	fine	fair
D	13%	medium	very poor
Jen-Sal	**13%**	**very fine**	**excellent**

maximum tissue concentration for dependable immunity

High tissue content of Jen-Sal Rabies Vaccine provides high vaccine virus titres for maximum immune response. In critical tests, dogs vaccinated with a standard 3 cc. dose of Jen-Sal vaccine consistently withstood virulent street virus challenge.

homogenized tissue for rapid reconstitution

Jen-Sal Rabies Vaccine reconstitutes in seconds. Tissue particles, minute enough to permit passage through a 24 gauge needle, assure a vaccine which is both effective and easy to administer. No evidence of undue pain or local irritation is noted on routine administration.

newest production refinements give you a superior product

Jen-Sal Rabies Vaccine contains living, safety-proved Flury virus modified by egg passage. Virus loss in storage is inhibited by new lyophilization refinements plus a Jen-Sal stabilizing agent. Meticulous control and rigorous testing guarantee a uniform vaccine of incomparable quality. Supplied in 10 dose bulk vial and 5—1 dose package.

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